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ERRATA.

Page 22, twelfth line from the bottom: for "feeble," read "slow."

Page 29, twenty-fourth line from top: for "In the cases," read "In other cases."

Page 58, tenth line from top: for "comparatively young," read "comparatively old;" eleventh line: for "quite old," read "quite young."

Page 66, third line from bottom: for "dura," read "brain."

Page 71, twenty-first line from bottom: for "cerebral apoplexia," read "chronic meningitis."

Page 95, third line from top: for "involuntary," read "voluntary."

Page 273: for "Lecture XLVII." read "Lecture XLVIII."

PREFACE.

These Lectures were delivered in the College of Physicians and Surgeons during the regular winter session of 1880–81, and were furnished to Student-subscribers, in parts, from time to time during the term, the last pages being distributed to the same within a week after the delivery of the final lecture.

It is but justice to Prof. Delafield, that I declare my personal responsibility for the accuracy of the following reports of his Lectures, as they have been prepared from my short-hand notes, and published without his revision.

The shouldering of so much responsibility, and the carrying out of a scheme within a short space of time which involved so much careful work, would never have been undertaken by me, had it not been for my enthusiastic appreciation of the great practical value of the Lectures, and the repeated and urgent requests of a large number of the Students and Alumni of the College.

M. JOSIAH ROBERTS.

New York, 4 West 28th Street,
May 23, 1881.

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GENTLEMEN: I have to ask your attention to some general considerations concerning inflammation. It is somewhat unfortunate that at the very beginning of the course I am obliged to present to you one of the most difficult subjects which occur during the whole of it, but it is impossible to avoid this. The moment we come to the consideration of the general diseases of the viscera we find that we come upon the consideration of inflammation, so that it is necessary at the outset to direct your attention to the subject of inflammation considered in its general aspect. In doing this, however, you must remember that we are considering the question as physicians and not as surgeons. The subject of inflammation, as it presents itself to the surgeon, is something quite different from the subject of inflammation as it presents itself to the physician. Surgeons are concerned with inflammation in connection with injuries, in the repair of wounds, in the destruction of tissue, and in the formation of abscess. Physicians, on the other hand, are concerned with inflammation of quite a different type. The inflammations we habitually see are not concerned in the repair of wounds or in the destruction of tissue, nor do we see a great deal of abscess, but we do see a great deal of inflammation in different parts of the body, and this inflammation produces medical symptoms.

We may have inflammation of the connective tissue; inflammation of the mucous membrane; inflammation of the different viscera of the body.

Connective tissue is composed of a basement-substance and of cells. The basement-substance, while alive, is nearly of a homogeneous consistency. After death, however, it presents the appearance of being made up of little fibres, and these fibres are again divided into smaller ones, called *fibrillæ*. These fibres are arranged in different ways in different parts of the body. Sometimes they are placed side by side, so as to form membranes of greater or less thickness. Sometimes they form small cords, such as make tendons. Sometimes the fibres are interlaced, as in subcutaneous tissue, and in the periosteum which covers bone. Sometimes the fibres are arranged so as to form a reticulum, such as we find in the intestines, lymphatic glands, and other parts of the body.

The cells which we find in connective tissue are of three kinds: First, we find round cells, which are identical in appearance with the white corpuscles of the blood. These are called moving or wandering cells. Besides these wandering cells we meet with fixed cells in the connective tissue, and these are of two kinds. Wherever there is a free surface we find it covered with flat, nucleated cells, that is, the cells have a very thin, flat body, and a nucleus which is thicker and more distinct. The edges of the cells are in close proximity to one another, so that the whole free surface is covered with these thin, flat cells. These are often called endothelial cells, for the reason that they resemble, to some extent, the epithelial cells, which we find upon mucous membranes. The third variety (also fixed cells) we find embedded in the substance of the connective tissue, and arranged in larger or smaller numbers between the fibres which make up the basement-substance. These cells will present themselves in different shapes, according to the different methods of preparation used for their demonstration. Sometimes they will look like narrow fusiform bodies; sometimes they are broad, long, and irregular cell like bodies with a distinct nucleus. At other times they occur as branching cells with very irregular bodies, and these cells contain one or several nuclei. Or again, if the demonstration be more perfect, you get small bodies anastomosing in all directions, and with one or more nuclei. This variety we may call the branching cells of the connective tissue. They can, as you see, hardly be said to have any regular shape. They are small, irregular masses of protoplasm which contain here and there one or more nuclei. In the tissue which is thus formed of basement-substance and of cells we have, in addition, blood-vessels, lymphatics, and nerves, these being distributed in greater or less numbers in the different portions of connective tissue which are situated in different parts of the body.

Now, the connective tissue may become inflamed in different ways, which we designate by names. We speak, *First.*—Of cellular inflammation. *Second.*—Of inflammation of the connective tissue with the production of pus, fibrin, and serum. *Third.*—Of necrotic inflammation. *Fourth.*—We speak of inflammation of the connective tissue with the production of pus, fibrin, and serum, and accompanied by the formation of new tissue. *Fifth.*—We have inflammation with the formation of abscess. *Sixth.*—Reparative inflammation. *Seventh.*—Hyperplastic inflammation. *Eighth.*—Tubercular inflammation. It is not, however, necessary for us to consider all the different varieties of inflammation. Abscess and reparative inflammation belong to surgery. Tubercular inflammation for other reasons I will not speak of at present.

CELLULAR INFLAMMATION, ACUTE AND CHRONIC.—In the first place we will consider simple inflammation. We mean by this a form of inflammation which is characterized by the production of new connective-tissue cells and of nothing else. There is no pus, no fibrin, no serum, the only inflammatory product being new connective-tissue cells. This form of inflammation occurs both as an acute and as a chronic process. The parts of the body where we meet with it most frequently are the pia mater and the peritoneum. There is both an acute and chronic meningitis, and an acute and chronic peritonitis, in which the only product of inflammation is new connective-tissue cells. There is no pus, fibrin, or serum, and yet the inflammation may be so severe as to cause the death of the patient. In the acute examples of simple inflammation we sometimes find a certain degree of congestion of the blood-vessels, and when this is the case the affected part looks redder than it should, for the reason that the blood-vessels contain an undue amount of blood; in other cases this congestion is absent. In chronic inflammation there is, as a rule, no congestion at all. The parts may, on the contrary, be rather paler than normal. The only change which can be observed by the naked eye is that the membrane loses its natural shiny and glistening appearance and becomes paler or a little swollen or thickened. This is all that you see. If, for example, it is the pia mater which is inflamed, when you make a post-mortem examination and are looking at the convexity of the brain, you will see no fibrin, no serum or pus, but upon closer examination it will be found that the pia mater, instead of being glistening in appearance, will be of the dull, sodden color, which I have already mentioned. If we examine the inflamed tissue minutely, however, with a microscope, we find that there are marked changes existing, and that these consist in the production of cells in very considerable numbers. These cells do not have the appearance of pus-cells, but resemble, more or less closely, the normal connective-tissue cells of this part. Besides these new cells which are produced we find cells which are swollen and more distinct than they are in common plain tissue. This is all we find in acute simple inflammation.

In the chronic forms of simple inflammation of connective tissue we find these same cells, but they occur in larger numbers and are larger in size than in acute inflammation. In a certain number of cases we find them grown together so as to form new membranes on the surface of the inflamed connective tissue, and it is in this way that adhesions form between the different parts of the peritoneum. These adhesions have the appearance of thin, delicate membranes, being composed of such cells as I have spoken of. This is what we find in the so-called simple chronic inflammation of connective tissue, the inflammatory product being only new connective-tissue cells.

INFLAMMATION WITH THE PRODUCTION OF PUS, FIBRIN, AND SERUM.—Now, in the second place, we come to inflammation with the production of fibrin, serum, and pus. There are regular changes in the blood-vessels. The blood-vessels contain more blood than they should, and the connective tissue is congested. Besides this, we find infiltrated in the substance of the tissue, also upon the surface, or collected in the cavities, pus, serum, and fibrin in different relative proportions. Sometimes there will be more serum, sometimes more fibrin, sometimes more pus. The relative proportion of these different inflammatory products varies very much in different cases. The earliest stages of this form of inflammation can be studied to good advantage on some of the transparent membranes of the lower animals. If, for example, we expose the mesentery of the frog in such a way that we can examine it under the microscope, we will find upon looking at the large arteries and the large veins, that they are occupied by a column of blood which is passing through them with considerable rapidity, and we distinguish at once two parts, a central and a peripheral portion. The central portion contains a column of red blood-corpuscles. The peripheral portion is formed of transparent plasma, and in this is seen a small number of white blood-corpuscles. If we look at the capillaries and smaller veins and arteries this regular arrangement of the blood is not observable. We can no longer distinguish a central and peripheral column. We find that the capillaries contain blood plasma, and in this are found white and red blood-corpuscles arranged irregularly. If now the part at which we are looking becomes inflamed from exposure to the air, or from the application of irritating substances, we find that the blood begins to circulate more slowly in the blood-vessels. In some of the blood-vessels it may even come to entire cessation, and then begin to move; but in all of the blood-vessels it will be found to circulate more slowly than normal. If now we direct our attention to the larger veins, we will see that the central column of blood remains the same, except, perhaps, it is somewhat denser and more compact from the presence of a larger number of red blood-corpuscles. In the peripheral portion we see an increased number of white blood-corpuscles, and as we continue to observe we see these becoming still more numerous; not only do they become more numerous, but they begin to adhere to the internal surface of the wall of the vein. If the process is continued for a sufficient length of time we will see in one of these veins that the peripheral layer of white blood-corpuscles is hardly moving at all, but that the central column continues to move with some rapidity, but not as rapidly as before the inflammatory process was begun. If we continue our observation we will see after a time that the external wall of the vein begins to present certain changes, that it is no longer smooth, but begins to look nodular, having little projections upon it. After a time we see, in addition to this, round cells entirely outside the vein. These are identical in appearance with the white blood-corpuscles which still remain in the interior of the vein. About this time we can see certain gross changes. The surface of the membrane is covered with a semi-transparent layer which can be scraped off, and if this be examined it will be found to consist partly of pus-cell, partly of coagulated fibrin, and partly of serum. If in addition to observing all this we turn our attention to the capillaries we will be enabled to see more exactly the way in which the white blood-corpuscles escape from the walls of the vessels. We will find a white blood-corpuscle passing slowly along through the vessel, and after a time this, in connection with one or two others, will adhere to the inner surface of the capillary for a time, and then moving on become adherent again. After it has been adherent for awhile we will begin to see a little knob on the outside of the capillary which gradually becomes larger, and the white globule on the inside of the vessel becomes smaller. We can now see one of these white globules with one part inside the vessel and one part outside of it. Finally, the globule which was inside the capillary will be found to be entirely outside of it. All these stages you can see very easily on account of the thinness of the wall of the capillary and the small number of blood-globules. We infer from what we see in the capillaries that the same thing takes place in the veins, but upon a larger scale than in the capillaries. The red blood-corpuscles are also capable of passing through the walls of the blood-vessels in the same way as the white. We infer, then, that the pus-globules, which we meet with in the inflammation of connective tissue, are nothing but migrated white blood-corpuscles. The fibrin which we find coagulated upon the surface or in the interstices of the connective tissue, seems to be derived partly from the plasma of the blood and partly from the white blood-globules. It does not exist in the plasma of the blood as such, but is formed by the union of certain substances which are contained in the plasma, with other substances which are contained in the white blood-globules. The serum seems to be derived from the plasma of the blood. You will infer then that the products of this kind of inflammation are derived from the blood-vessels. You will see also the difference between this and simple inflammation. In simple inflammation the blood-vessels take very little share in the inflammatory process, the principal participant being the connective-tissue cells. In the second form of inflammation the connective-tissue cells do not share at all in the inflammatory process, but it is the blood-vessels that are concerned. It is from the blood circulating in the blood-vessels that the characteristic products of pus, fibrin, and serum are derived.

Pus in Medical and in Surgical Inflammation.—When I speak of pus in this connection, however, you must not think of it as you would in thinking of inflammation from the surgical point of view. Pus presents itself to the physician in a different way from that in which it presents itself to the surgeon. For example: in ordinary lobar pneumonia we find the air-vesicles filled with pus-globules and fibrin, while the walls of the air-cells are in a normal condition. The lung under such circumstances is perfectly hard and firm, and this is the way that pus presents itself to us in that form of inflammation of which I am speaking.

Suppose, on the other hand, you have an abscess of equal size with one of the lobes of the lung. Such an abscess instead of being hard will have a large cavity filled with pus and its walls will present decided changes. In the case of pneumonia we have the deposit of a dry substance without the tissue undergoing any change. In the other case we have a large cavity filled with a thick yellow fluid, and the walls of this cavity present very decided changes.

With the conditions which produce abscess we have nothing to do. I merely present the subject to you in this way for the purpose of explanation. The sort of pus I am speaking of is the result of medical inflammation. It is pus which does not as a rule go on to the production of abscess, but which occurs in what we may call a dry form. When it occurs in a fluid form, as in the pleura, it is fluid simply because the pus-globules are suspended in the serum which comes from the blood-vessels. It is very important to keep in mind what is understood by pus in surgical inflammation and what is understood by pus in medical inflammation.

Removal of Inflammatory Products.—After the pus, serum, and fibrin are produced, one of two things happens. The patient may die, and after death we may find upon examination that the pus, serum, and fibrin still exist. Or if, instead of dying, the patient should get well, this pus, fibrin, and serum will disappear, and the particular parts which were the seat of the inflammatory process will return to their normal condition. The serum disappears first, but the fibrin and pus have to degenerate and become converted into granular matter before they can be absorbed so as to leave the tissue in the same condition it was before it was inflamed. We see this, for example, in the lungs. If a patient recovers from an attack of lobar pneumonia the fibrin and pus is either absorbed or expectorated ; a very large part is absorbed, however. After this the air-vesicle is left in its normal condition. We can understand how it is that the parts can return to their normal condition when we remember that the connective tissue takes no part whatever in the inflammatory process, so that when a patient recovers from the inflammation, all that is necessary is that the inflammatory product be absorbed by the lymphatics. The connective tissue remains almost passive during the entire process. In the other case, however, these inflammatory products are not absorbed, but they remain and they may remain for a long time. They may remain as pus, fibrin, or serum, or in other cases they undergo degenerative changes, the fibrin or pus becoming converted partly into fatty, partly into granular matter, and in this way we have the products of inflammation left behind for a long time.

LECTURE II.

INFLAMMATIONS OF CONNECTIVE TISSUES—Concluded.—INFLAMMATIONS OF MUCOUS MEMBRANES.

Necrotic Inflammation.—Inflammation with the Production of Pus, Fibrin, Serum, and new Connective-Tissue Cells.—Hyperplastic Inflammation.—Structure of Mucous Membranes.—Catarrhal Inflammation of Mucous Membranes, Acute and Chronic.—Purulent Inflammation of Mucous Membranes, Acute and Chronic.—Croupous Inflammation of Mucous Membranes.

Gentlemen : We were speaking at our last lecture of the forms of inflammation, and considered—first, cellular inflammation ; second, inflammation of the connective tissue with the production of pus, fibrin, and serum.

Necrotic Inflammation.—To-day we will go on to the consideration of necrotic inflammation, inflammation accompanied with death of the tissues. This form of inflammation is simply a modification of the one we were considering at the close of the last lecture. I told you that in inflammation accompanied with the development of fibrin, serum, and pus, the blood-vessels contained more blood than they should, but that the blood still continued to circulate. If it were not for the continuance of the circulation of the blood, the migration of the white blood-globules would not take place, nor would we have the production of pus. Sometimes, however, circulation in the vessels ceases and they become engorged with blood. If this condition only lasts for a short time the nutrition of the parts supplied by the engorged vessels may not be seriously interfered with ; but should it continue for a considerable length of time, the same condition results as would be induced by the ligation of the artery and vein going to the part, namely, the tissues die. After death there are various degenerative changes which result in an absolute loss of tissue. This is what is meant by *necrotic inflammation*, a form of inflammation in which the blood-vessels are principally concerned, and in which the tissues are affected by the blood-vessels becoming engorged with blood and the circulation arrested.

INFLAMMATION WITH PRODUCTION OF PUS, FIBRIN, SERUM, AND NEW CONNECTIVE-TISSUE CELLS.—The fourth variety of inflammation which we have to consider consists of a combination of two of the forms of inflammation already discussed. In this form of inflammation there is a production of serum, fibrin, and pus, and at the same time there is also produced new connective-tissue cells. In a certain number of examples these changes are not exactly simultaneous ; serum, pus, and fibrin are first produced, and new connective-tissue cells somewhat later. If the patient recovers, the serum, pus, and fibrin are absorbed, but the tissues do not return to their former condition for the reason that the new connective-tissue cells are not absorbed. In this form of inflammation both the blood-vessels and the connective tissue are involved.

HYPERPLASTIC INFLAMMATION.—Lastly, we have a variety of inflammation which we may call *hyperplastic*. There is no production of fibrin, pus, or serum. Connective-tissue cells are produced, but not in very great numbers ; in addition there is a production of basement-substance. If it is the peritoneum which is the seat of this form of inflammation, it may sometimes be thickened to the extent of half an inch. The cells produced in the early stages of this variety of inflammation are true connective-tissue cells, and require no subsequent transformation to become connective-tissue elements. The whole peritoneum becomes thicker in consequence of the production of cells and of basement substance, the cells being somewhat more numerous than in ordinary connective tissue, but still not very numerous. These make up the different forms of inflammation of connective tissue which we will meet with when we come to the consideration of the diseases of the different parts of the body.

INFLAMMATIONS OF MUCOUS MEMBRANES.

STRUCTURE OF MUCOUS MEMBRANES.—We now come to the inflammations which affect mucous membranes. All mucous membranes of the body consist essentially of three parts : the *stroma*, which forms the framework, and the *epithelial cells* covering it, together with the *glands* imbedded in the substance of the stroma. The stroma is sometimes arranged so as to form nearly a flat surface, but more often it forms an irregular surface in the shape of papillæ, situated at regular intervals, and covered with epithelial cells peculiar to mucous membrane. The spaces between the papillæ are filled with epithelial cells so that the surface of the mucous membrane is flat, although the stroma is rough and hilly. Sometimes these cells are flat, and are spoken of as *pavement cells ;* sometimes *ciliated*, sometimes of a *cuboidal* shape. The glands of the mucous membrane are either *straight follicles* or *racemose* glands ; and in either case they open upon the surface of the mucous membrane. The function of these glands is to produce a substance called *mucus*. The stroma of the mucous membrane is ramified by blood-vessels, which supply nourishment to the membrane; there are also found in the stroma, lymphatics, which absorb whatever there is to be absorbed.

While the mucous membrane is in its normal condition there is a constant growth and production of epithelial cells covering its surface. The most superficial cells are continually being removed from the surface of the part, and their place filled by new cells which are formed to replace the old ones. If, for example, the mucous membrane of the mouth be in a healthy condition, and you examine the saliva with the microscope, you will find epithelial cells in it which were desquamated from the surface of the mucous membrane; this is a normal process, and it takes place slowly. In a healthy mucous membrane the glands are constantly producing mucus, but only in sufficient quantity to keep the surface of the membrane properly lubricated.

The forms of inflammations that we find affecting mucous membranes, are, catarrhal, purulent, croupous, and gangrenous inflammations.

ACUTE CATARRHAL INFLAMMATION OF MUCOUS MEMBRANE.—In the first place, of catarrhal inflammation. Catarrhal inflammation of mucous membranes occur both as an acute and chronic process. In the acute form we find the affected mucous membrane congested, which gives to it a brighter red color. There is in many cases a certain amount of swelling, and at the very beginning of the process the mucous membrane is dryer than it should be, on account of less mucus being produced by the glands. After the inflammation has lasted for a little time the functional activity of the mucous glands is greater, and the surface of the membrane is bathed with an increased amount of mucus. Mucus produced in too great quantity does not have the consistency or qualities of normal mucus; it is sometimes thicker, sometimes thinner, and is acrid and irritating to the parts. The degree of congestion varies considerably in different cases, and is regulated by the intensity of the inflammatory process. If the inflammation be very severe the parts will be very much congested, the blood-vessels will be gorged with blood, and the color of the mucous membrane, instead of being bright red, will be of a darker, bluish, livid color. In milder cases the mucous membrane will be of a redder color than it should be, and the epithelium will also undergo changes. The process of desquamation of the superficial epithelial cells, and the production of new epithelial cells goes on more rapidly, so that an examination of the mucus taken from the inflamed surface would reveal an increased number of epithelial cells. Sometimes the process of desquamation goes on so rapidly, that new cells are not formed fast enough to take the place of the old ones, and we have as a result superficial ulcerations.

The changes which take place in acute catarrhal inflammation are always much more evident during the life of the patient than after death, and you will observe that these changes are not, properly speaking, structural changes. The blood-vessels contain more blood, the mucous glands have their function altered, there is swelling of the mucous membrane, and there is increased desquamation of the epithelial cells ; but, unless this latter process is sufficiently marked to produce ulceration, there are no structural changes in the mucous membrane. Hence, we may examine the part after death, and find no changes indicating that inflammation existed during the life of the patient. This is seen frequently enough at the autopsies of patients in whom we have observed before death that conjunctivitis was still going on. Upon examination of the eye after death it will be observed that the congestion has disappeared, the swelling has gone down, and there is perhaps no mucus present. It is important for you to remember this fact, that acute catarrhal inflammation cannot be affirmed or denied from evidence obtained at an autopsy.

CHRONIC CATARRHAL INFLAMMATION OF MUCOUS MEMBRANES.—In chronic catarrhal inflammation of mucous membranes, structural changes do take place and persist after the death of the patient, and these changes can be as readily demonstrated after death as during life. In a certain number of the examples of chronic catarrhal inflammation we find the mucous membrane congested, and in addition we find the blood-vessels larger than they should be and more numerous. In other examples we find exactly the opposite condition. The blood-vessels contain less blood than they should, and the mucous membrane, instead of being redder than normal, is of a white, grayish, or sodden appearance. The blood-vessels, instead of being more numerous and dilated, may appear to be less numerous than the blood-vessels in normal mucous membrane. The functional activity of the mucous glands is always affected in chronic catarrhal inflammation, but here again in different examples we find the mucous glands affected in two different ways. In a certain number of cases the production of mucus will be increased, and this mucus will be of an abnormal character. In other cases, exactly the opposite condition obtains. There is too little mucus produced, and as a result of this, the mucous membrane is constantly dry, and of a peculiar shining appearance. In ordinary naso-pharyngeal catarrh, you have a very good example of both these changes. There are a certain number of these cases in which there is too much mucus ; the patients are continually blowing their nose, and raising mucus from their throat, and, upon inspection, strings of mucus will be found hanging from the walls of the pharynx. In other cases, the mucous membrane of the pharynx looks perfectly dry, there being no mucus whatever upon it. This same variety of inflammation affects other mucous membranes.

Certain changes take place in the stroma of the mucous membrane : it is sometimes hypertrophied, sometimes atrophied. If it is hypertrophied, the hypertrophy may be either uniform or circumscribed ; if circumscribed, the hypertrophies form little tumors projecting from the surface of the mucous membrane. The mucous glands may be also hypertrophied or atrophied, and here again the hypertrophy may involve all of the glands of the affected mucous membrane, or it may be confined to a limited number of the glands, and the rest remain in a normal condition. In still other cases we find circumscribed hypertrophies of stroma and circumscribed hypertrophies of glands, accompanied by the production of little polypoid growths. In still other cases the glands become cystic; the ducts of the glands become narrowed or obstructed, and there is an accumulation of mucus in the lower part of the glands, which results in their distention, so as to form cysts, sometimes of considerable size—large enough to be seen with the naked eye. These changes which we have described, you observe, are sufficient to produce well-marked structural changes in the mucous membrane. If the blood-vessels are increased in number and size, if the stroma is atrophied or hypertrophied, if the mucous glands are atrophied or hypertrophied, or cystic, all these changes can be recognized as well after the patient's death as during his life. I should have stated, that in this form of inflammation we have the same changes taking place in the epithelium as in acute inflammation, namely, desquamation of the epithelial cells. The desquamation may be so rapid as to give rise to superficial ulceration.

PURULENT INFLAMMATION OF MUCOUS MEMBRANES.—Purulent inflammation of mucous membranes is merely a modified condition of catarrhal inflammation. It may occur either as an acute or chronic process, and in either case we have the same changes that are found in acute or chronic catarrhal inflammation of mucous membrane; but in addition to these changes there is a production of pus-cells. These pus-cells are produced both on the surface of the inflamed membrane and in its substance, so that during the life of the patient the increased amount of mucus which covers the part contains pus-cells, and at the same time the stroma of the mucous membrane is infiltrated with them. There are different degrees of purulent inflammation. In some cases there are only a few pus-cells, and these you can call catarrhal or purulent as you choose, for they are upon the border-line between catarrhal and purulent inflammation. In other cases the amount of pus produced will be very large, and the infiltration of the tissues with pus may be very considerable. When this is the case the inflammation is already much more severe, and the local and constitutional symptoms are much more marked than if the patient were suffering from catarrhal inflammation. These two forms of inflammation pass by gradual degrees from one into the other, the only difference between them being that while pus is present in one it is absent in the other.

CROUPOUS INFLAMMATION OF MUCOUS MEMBRANES.—Now we come to croupous inflammation of mucous membranes, or, as it is sometimes called, diphtheritic inflammation, both names being used to designate the same anatomical form of inflammation. In croupous inflammation we find the mucous membrane congested to a greater extent, as a rule, than in catarrhal inflammation. The mucous membrane is either of a red color or of a bluish, livid color, according to the amount of congestion. Swelling of the mucous membrane is usually more marked than in cases of catarrhal inflammation. Besides the congestion and the swelling, we find the surface of the mucous membrane covered with a layer of new material—with what is called *false membrane*. and this is composed of fibrin. of pus. of epithelial cells. and very often of bacteria. The fibrin is coagulated, either in the form of a network or of granular matter, or in the form of irregular amorphous masses. The fibrin varies in quantity with the severity of the inflammation. Sometimes it forms a very thick layer, sometimes a very thin layer. so thin as not to be easily seen with the naked eye. The pus-cells look like ordinary pus-cells—look like the ordinary white globules of the blood. These remain in place for a considerable length of time. then enlarge and undergo fatty degeneration. A certain number of the epithelial cells resemble those of the mucous membrane : others are changed so as to have somewhat the appearance of coagulated fibrin. and still others are changed into irregular branching fibres. which no longer resemble epithelial cells. The bacteria are usually in the form of what are called *micrococci* ; that is, minute granulations collected into little masses. Less frequently we find what are called *rod-shaped* bacteria ; that is, they have the form of little rods.

Besides the production of fibrin and of pus, which forms the false membrane upon the surface of the inflamed mucous membrane, we have also an infiltration of fibrin and pus into the substance of the inflamed tissue. Sometimes the fibrin and pus are deposited between the layer of epithelial cells and the surface of the stroma, so that the epithelial cells are separated from the stroma by this new layer of pus and fibrin. Sometimes the pus and fibrin are infiltrated into the interstices of the stroma, the extent of the infiltration depending in all cases upon the severity of the inflammation, as does the amount of pus and fibrin produced.

In a certain number of cases the accumulation of pus and fibrin will be so extensive as to compress the blood-vessels, while at the same time some of the blood-vessels are blocked with blood which ceases to circulate. When this is the case a portion of the mucous membrane will die, degenerate, and slough away. It is in this manner that ulcers are formed, and vary in depth according to the extent of the infiltration of the tissues by pus and fibrin, and the extent to which the blood-vessels are plugged with stagnant blood. Thus in many cases of that form of intestinal inflammation known as dysentery, ulcerations take place which are sometimes quite superficial and in other cases very deep. In still other cases these ulcers will extend to the peritoneum, and in the worst cases involve the peritoneum itself.

LECTURE III.

INFLAMMATIONS OF MUCOUS MEMBRANES —CONCLUDED.—INFLAMMATIONS OF THE VISCERA.

Gangrenous Inflammation of Mucous Membranes.—Conditions Favorable to the Development of Gangrenous Inflammation.—Anatomical Elements of the Viscera.—Congestion of the Viscera.—Parenchymatous Inflammation of the Viscera, Acute and Chronic.—Interstitial Inflammation of the Viscera, Acute and Chronic.—Diffuse Inflammation of the Viscera, Acute and Chronic.—Recapitulation.—Occurrence of Inflammations of Connective Tissues, Mucous Membranes, and the Viscera.

GANGRENOUS INFLAMMATION OF MUCOUS MEMBRANE.—*Gentlemen:* To-day we will begin with the consideration of that form of inflammation of mucous membranes known as *gangrenous inflammation*, by which it is meant, that a portion of the affected tissue dies and sloughs away. In a certain number of the cases a portion of the tissue simply dies and does not become detached from the surrounding living portions of mucous membrane, nor does it undergo decomposition. In other cases, however, these portions of tissue not only die, but they putrefy; bacteria are formed, and there is an evolution of gases peculiar to the process of putrefaction.

Causes.—Gangrenous inflammation of mucous membranes usually occurs under one of three conditions, although sometimes two or more of these conditions exist in the same case. If congestion occurs and goes on to complete stagnation, then gangrene will take place simply as a result of this. The extent of tissue involved varies in different cases, from a very slight amount to the whole of the inflamed mucous membrane. There are some cases of dysentery in which one or two feet of the large intestine will become gangrenous in this way. Another way in which we have gangrenous inflammation taking place is that, when croupous inflammation is going on, the amount of pus and fibrin produced and the infiltration into the inflamed membrane may be so great as to cause undue pressure upon the tissue, and, when this is the case, these portions die and slough away. Under such circumstances the portions of involved tissue are not very large.

Besides these two conditions which lead to gangrenous inflammation, we find that there are some epidemics of inflammations of mucous membrane in which there is a tendency to gangrene. What the reason of this is we do not know, but in a considerable number of cases there will be gangrene and sloughing of a larger or smaller portion of mucous membrane. These, then, are three ways in which we may have gangrenous inflammation taking place.

INFLAMMATIONS OF THE VISCERA.

Anatomical Elements of the Viscera.—The viscera of the body consist essentially of three anatomical elements : of stroma, of blood-vessels, and of cells. The stroma forms the framework, the blood-vessels furnish the nourishment, and the cells perform the function of the organ in which they exist. In addition to these we find nerves and lymphatics ramifying the viscera, but the essential parts to be remembered are the stroma, blood-vessels, and cells. The cells differ very much from those of the connective tissue, and from the epithelial cells covering mucous membranes, of which we have already had occasion to speak. The cells of connective tissue perform no function except that of living. The same is the case with the epithelial cells covering mucous membranes; they live and perform no other function, except, perhaps, they may help to produce mucus. The cells of the viscera, however, not only exist, but they perform the function of the organ in which they exist. Thus, in the liver, the peculiar cells not only are alive, but, in order that the regular processes of the body may be carried on, these cells are constantly performing a certain function. The same is the case with the cells of the kidneys, of the spleen, of the pancreas, and of the other viscera of the body.

Congestion of the Viscera.—This difference in the character of the cells makes at once a new set of symptoms when we come to inflammation of the viscera ; for when a viscus is inflamed the function of its cells is interfered with, and consequently the functions of the viscus itself are interfered with. When we come to consider inflammations of the viscera, we have to consider simple congestion, and parenchymatous, interstitial, and diffuse inflammations.

If a mucous membrane becomes congested, or a portion of connective tissue becomes congested, except for the discomfort caused thereby, no great harm is done ; but in congestion of the viscera much more marked symptoms are produced. In visceral congestions there are no structural changes ; the stroma remains the same, the cells are not changed, except, perhaps, they may be a little swollen, and the blood-vessels show no alteration other than that they contain more blood than they should. Hence it is that congestion of the viscera may exist during life and no signs of its existence be discoverable after death. The congestion of a viscus may be so severe as to produce marked symptoms, or even death, and yet at the autopsy the particular organ will appear perfectly normal. This statement finds illustration in congestions of the brain and spinal cord. Again, if the liver be congested, there will be an interference with the processes of digestion, and yet, after death, no changes be found in that organ to indicate that it had ever been congested. The same is true of the kidneys. Congestion of the kidneys produces changes in the urine and general constitutional symptoms. In this particular example, however, congestion of the viscus usually persists after death, and may be recognized at the autopsy.

PARENCHYMATOUS INFLAMMATION OF THE VISCERA.—By parenchymatous inflammation of a viscus we mean that form of inflammation in which the stroma and the blood-vessels are not changed. But the cells of the viscus, those structures which perform the function of the organ to which they belong, do undergo certain regular changes. The first of these consists in a swelling of the cells and in a change of the substance of which the cells are composed. Suppose a case of parenchymatous inflammation of the cells of the liver. These cells are of a polygonal form, and are furnished with a nucleus. The substance which they contain is not homogeneous, but it is transparent. The first change noticeable in inflammation of these cells is that they become larger, and the substance which they contain is no longer as transparent as in the normal liver cell; it begins to show fine granulations mixed with the cell-body. Then, as the congestion advances, the cell-body becomes thick and sufficiently opaque to obscure the nucleus, and the appearance of the granules in the cell-body becomes still more marked. These granules are apparently of an albuminous nature. As the process goes on the cell-body becomes still more coarsely granular, and mixed with this we begin to have little drops of fat, which can be dissolved by chloroform or ether; the nucleus may be entirely obscured by the granules and by the accumulation of fat in the cell-body. Finally, the cells become a mass of fat and granules, and no longer possess a distinct shape. These are the changes which take place in parenchymatous inflammation of the viscera, and which lead to the destruction of the cells, if the inflammatory process be sufficiently intense and of sufficiently long duration. Sometimes patients die in the early stages of parenchymatous inflammation, sometimes in the last stages, and between these two we get different gradations of structural changes in the cells.

ACUTE PARENCHYMATOUS INFLAMMATION.—Parenchymatous inflammation may run an acute or chronic course, may last but a few days or may last weeks, months, or years. If it only lasts for a few days, and the patient survives the immediate effect of the inflammation, then the viscus will return to a normal condition. Exactly how this takes place we do not know, but we have to suppose that the cells which are originally affected are capable of returning to their natural condition, or that they are replaced by new normal cells. At all events we know that a patient may suffer from parenchymatous inflammation, and, if he recovers, after a time the viscus will perform its function as well as ever.

CHRONIC PARENCHYMATOUS INFLAMMATION.—Chronic parenchymatous inflammation is not so apt to be followed by complete recovery of the affected viscera as the acute form, but still in this case also the viscus apparently returns to a natural condition and performs its function as well as ever. This is an important point in the clinical history of parenchymatous inflammations, and it is one reason why, in practice, we should distinguish between this and other varieties of inflammation, for the prognosis varies in different forms of inflammation.

ACUTE INTERSTITIAL INFLAMMATION OF THE VISCERA.—In interstitial inflammation of the viscera it is the connective-tissue stroma which is involved. The inflammatory process may be either acute or chronic. If acute, it is usually suppurative—inflammation with the production of pus. The production of pus may be either diffuse or circumscribed; more frequently it is circumscribed, occasionally it is diffuse. In this form of inflammation you will find the stroma either in foci, or through a considerable area of the viscus, infiltrated with pus-cells. If these pus-cells are not very numerous, the stroma will be simply swollen, but if they exist in large numbers a little abscess will be formed. The cells of the viscus usually undergo parenchymatous or degenerative changes as an accompaniment or result of the suppurative inflammation of the stroma. If the inflammation involves a considerable portion of the stroma of the viscus, the cells will disintegrate, and in this way cavities will be formed and filled with a fluid which resembles pus. This fluid is composed partly of serum, partly of pus-globules, partly of the cells of the organ, and partly of broken-down fragments of the viscus. In this way we sometimes have cavities formed of considerable size, and these cavities acquire a wall.

CHRONIC INTERSTITIAL INFLAMMATION OF THE VISCERA.—In chronic interstitial inflammation of the viscera we do not have pus formed, as a rule, but simply have the formation of new connective tissue, which is, from the beginning, composed of cells and basement-substance. There is not at first a production of basement-substance, but there is, from the beginning, a new growth of connective tissue like the original stroma of the viscus, and differing from it only by having an increased number of cells. One of the best examples of this form of inflammation is found in cirrhosis of the liver, where we have not a simple production of connective-tissue cells, but new connective tissue, consisting of basement-substance and connective-tissue cells. In both acute and chronic interstitial inflammation the changes, you observe, are structural ones, and from which the patient cannot entirely recover. If a patient suffer from acute interstitial inflammation of a viscus with the production of pus, it never returns to a normal condition, although the patient may get well. The same is true of chronic interstitial inflammation; the new tissue formed during the inflammatory process being permanent. Again, a knowledge of this fact is important from a clinical view; it is important to differentiate between interstitial and parenchymatous inflammations. In parenchymatous inflammations the affected viscus may return to its normal condition, but if affected by interstitial inflammation, it can never return to its normal condition.

ACUTE DIFFUSE INFLAMMATION OF THE VISCERA.—The diffuse inflammations of the viscera differ from the inflammations of which we have just spoken in the fact that all of the constituents of the affected viscus are involved in the inflammatory process—blood-vessels, stroma, and cells. Diffuse inflammation may run an acute or chronic course. If it runs an acute course, the changes which we find in the viscus are these: the viscus is somewhat increased in size—it is swollen, the blood-vessels contain more blood than normal—they are congested, and the stroma is infiltrated with serum and pus-cells. The cells of the viscus are swollen and more or less degenerated. The degree to which these changes are found varies in different examples of diffuse inflammation; they are sometimes marked, sometimes comparatively slight. The viscus may be swollen to double its normal size; it may be of a livid or purplish color, and, upon examination after death, we may find the stroma infiltrated with serum and pus-cells, degeneration having taken place. In other cases the changes are less marked; the swelling is slight, there is only moderate congestion of the blood-vessels, the cells have undergone very little change, and the amount of serum in the stroma is not large. According to the intensity of the inflammation so will be the character of the symptoms during life.

Acute diffuse inflammation is again a form of inflammation from which the patient may entirely recover, if he does not die from the immediate effects of the inflammation. This, however, does not always take place; the patient may not die, and yet may not entirely recover. When this happens, it is the result of acute diffuse inflammation being followed by chronic diffuse inflammation. One of three conditions takes place when a viscus is the seat of acute diffuse inflammation: the patient dies, or recovers his normal condition, or the acute passes into the chronic form of diffuse inflammation.

CHRONIC DIFFUSE INFLAMMATION OF THE VISCERA.—In chronic diffuse inflammation of a viscus we find, as before, all the constituents of the organ involved—stroma, blood-vessels, cells. The stroma is increased in amount, partly by the production of permanent new connective tissue, and partly by the infiltration with enlarged cells. Some of these cells are apparently pus-globules, some of them new connective-tissue cells. The blood-vessels also undergo changes, not only in the way of congestion, but their walls may undergo changes, and the calibre of the lumen of the vessel may become narrower. The cells of the viscus undergo degenerative and destructive changes. A chronic diffuse inflammation of a viscus once begun, never entirely ceases; the organ never returns to a condition of health. The inflammatory changes go on, and the best that can be hoped for in such a patient is, that the periods of intermission may be protracted as long as possible; sometimes these periods of intermission may last for years, sometimes for a few months, and sometimes only for a few weeks, and then the inflammatory process will go on again.

RECAPITULATION.—You see now the difference between these various clinical conditions of the viscera. We may have congestion taking place without producing structural change in the organ; parenchymatous inflammation running an acute or chronic course; the viscus returning to its natural condition, if the patient recovers; interstitial inflammation, in which, if the patient recovers, the viscus does not return to a natural condition; diffuse inflammation running an acute and chronic course; in the acute form, if the patient recovers, the viscus may return to a natural condition, or the acute process may be succeeded by the chronic; in chronic diffuse inflammation the viscus does not return to a normal condition, and the inflammation has a tendency not to stop.

OCCURRENCE OF INFLAMMATIONS OF CONNECTIVE TISSUES, MUCOUS MEMBRANES, AND THE VISCERA.—These different forms of inflammation of which we have spoken—inflammations of connective tissues, of mucous membranes, and of the viscera, present themselves to us under two different conditions. They may either occur as local inflammations, or they may occur as inflammatory complications, or as a characteristic part of general diseases: this distinction is very important for you to remember.

Thus, for example, in idiopathic cerebro-spinal meningitis you have a general disease, but this general disease is always accompanied by a characteristic inflammatory lesion; in this particular case the lesion is of the brain and spinal cord, and the symptoms from which the patient suffers will be due to a combination of these two conditions. He will suffer partly from symptoms due to the poison of the disease, and partly from symptoms due to local inflammation. The same may be said of diphtheria, typhoid fever, and other general diseases which have characteristic local lesions. The symptoms will partake of the double character of the disease, there will be symptoms belonging to the general character of the disease, and certain symptoms which belong to the local lesion.

Then, on the other hand, we have local inflammations which do not belong to general diseases. If a patient have simple tonsillitis, he suffers merely from local inflammation of the tonsils. There are, as a rule, both local symptoms and general symptoms, but the general symptoms are dependent entirely upon the local inflammation. One or both tonsils will be swollen, which will give rise to more or less pain. There will be present the characteristic symptoms of inflammation, swelling, heat, pain, and congestion. In addition to these, however, the patient will usually have a febrile movement, which may be preceded by rigor. So here also, the patient will suffer from two distinct sets of symptoms, but the constitutional symptoms will be due to the local lesion. This distinction you will find, as I have said, an important one to bear in mind: that the different inflammations of connective tissue, of mucous membranes, and of the viscera, may occur as local inflammations, or inflammatory complications of general diseases. When they occur as local inflammations, you have both local and general symptoms dependent upon the local lesion. When they occur as inflammatory complications of general diseases, you have two distinct sets of symptoms, one dependent upon the local lesion, the other dependent upon the general disease.

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LECTURE IV.

INFLAMMATIONS OF THE MEMBRANES OF THE BRAIN.—PACHYMENINGITIS.

Inflammations of the Duramater.—Anatomy and Relationships of the Duramater.—Forms of Duramater Inflammation.—Acute Pachymeningitis.—Thrombosis of the Venous Sinuses of the Duramater.—Chronic Pachymeningitis.

GENTLEMEN: We begin to-day the consideration of the inflammations of the membranes of the brain. These membranes are two in number, the *duramater* and the *piamater.*

We have to consider first the inflammations of the duramater. These inflammations are not very common, but when they do occur they are apt to give rise to well-marked symptoms, and not infrequently they prove fatal.

ANATOMY AND RELATIONSHIPS OF THE DURAMATER.—The duramater is a dense, fibrous, connective-tissue membrane, and is related both to the bones of the skull and to the brain. So far as the bones of the skull are concerned the duramater acts as an internal periosteum. It forms an essential part of the bones of the skull, just as the periosteum does to the other bones of the body, and the vessels from its external surface pass into the bones of the skull just as vessels in the periosteum elsewhere pass into the bones. On its inner surface, however, the duramater serves as one of the coverings of the brain, and it is provided with a free surface covered with a layer of endothelial cells. Anatomists are in the habit of saying that there also exists such a structure as the *arachnoid membrane,* and describe it as a closed serous sac having visceral and parietal layers. This may be true in an anatomical point of view, but so far as pathological processes are concerned, an arachnoid membrane does not exist; all we have to consider are the duramater and the piamater. We have in the duramater certain large veins or channels, which are called *the venous sinuses.* These are of great size, and their function is to receive the blood from the veins and conduct it to the internal jugular veins. These sinuses are very apt to become involved in inflammations of the duramater. So you have to remember these three relationships of the duramater:

its relation to the bone on the outside, to the piamater on the inside, and that embedded in it are large venous sinuses.

Forms of Duramater Inflammations.—The forms of inflammation which we find occurring in the duramater, are acute inflammation, chronic inflammation, and syphilitic inflammation. It is customary to speak of all the inflammations of the dura under the name of *pachymeningitis*; so we speak of acute, chronic, and syphilitic pachymeningitis.

The inflammations of the piamater are usually spoken of under the name of *meningitis*, and it is the inflammation of this membrane which is referred to when the term meningitis is used without qualification.

Acute Pachymeningitis.—Acute pachymeningitis may begin and involve either the external layer of the duramater, the internal layer, or both, and the symptoms accompanying such an inflammation will differ according to which portion of the membrane is affected.

Inflanimations of the external layer of the duramater are usually secondary. We find it secondary to inflammations of the periosteum which covers the outside of the cranial bones, to fractures of the cranial bones, to inflammatory processes which affect the cranial bones, and to suppurative inflammations of the orbit of the eye. If the patient dies the lesion which we find after death is an accumulation of pus between the duramater and skull bones. The extent of this accumulation will vary with the extent to which the duramater is involved. These collections of pus are usually circumscribed, for inflammations of the duramater are not diffuse to any extent. The symptoms produced by this condition are not marked, and consist principally of local pain, usually referred to that part of the head where the inflammation is going on in the duramater. There may also be a little febrile movement; there are no cerebral symptoms practically speaking.

The principal danger in this form of inflammation is that the process may extend from the outer and involve the inner surface of the duramater. When this happens we do have very marked symptoms.

Inflammations of the internal layer of the duramater may be secondary to inflammation in the external layer, or we may find it as a complication of pyæmia, as a complication of puerperal fever, or as a complication of diffuse nephritis—chronic Bright's disease. It also occurs in connection with small-pox and scarlet fever. Occasionally it is also consequent upon erysipelas of the face and scalp. It is also possible for it to occur as an idiopathic disease. If the patient dies from this inflammation, and he is very apt indeed to do so, after death we find the inner surface of the duramater covered with a layer of pus and fibrin which may be either diffuse, or circumscribed. The substance of the duramater is usually not very much changed. It may be a little thickened, the vessels may be somewhat congested, and there may be a moderate production of new connective-tissue cells and pus-cells, but the bulk of the inflammatory product is upon the free surface of the duramater, forming a sort of false membrane which can be detached. If the inflammation is sufficiently intense it extends to the piamater, and this also becomes inflamed, so that the patient suffers at the same time from pachymeningitis and meningitis proper.

Thrombosis of the Venous Sinuses.—Secondary changes in the venous sinuses of the duramater are very apt indeed to take place in this form of inflammation, the changes consisting of an accumulation of blood in these sinuses—thrombi as they are called. The formation of these thrombi adds to the danger of the disease, for they are likely to degenerate and their detached particles pass down through the veins into the right auricle, from the right auricle into the right ventricle, and from the right ventricle through the pulmonary artery into the lungs, where they become embedded and give rise to lesions which add to the dangers of the patient. Not infrequently do we meet with cases which apparently would have recovered had it not been for the occurrence of these secondary lesions, or thrombi, and the detachment and lodgement of portions of these thrombi.

Symptoms.—The symptoms of this form of inflammation of the duramater are usually pretty well marked. The lodgment of portions of a detached thrombus in some of the viscera is often attended with rigor. There is a febrile movement which is usually fairly marked without running very high, the temperature varying from 100° to 103° or 104°; it is not apt to run over this. The temperature has no characteristic rise and fall; sometimes it is a little higher, sometimes a little lower, but whatever variation there is in temperature does not necessarily mark the progress of the disease. The temperature may be lower when doubtless the patient is very much worse. The pulse is usually at first rapid, then not so rapid, and toward the close of the disease it may become slow and very infrequent. Again it may be about normal, or a little more rapid than normal, full and pretty tense. After the disease has lasted for two or three days, the pulse, in a considerable number of the cases, becomes slow; it may fall to seventy, sixty, and even forty to the minute, being at the same time a pretty full pulse. As the disease goes on, and especially if it is approaching a fatal termination, the pulse is very apt to run up to 120, 140, or 150, and at the same time become very feeble. The co-existence of a feeble pulse and elevated temperature is always of help in making a diagnosis of inflammation of any of the membranes of the brain, and is considered quite characteristic. There is a period in this disease, with a temperature of 103° or 104°, accompanied by a pulse of only fifty or sixty, which is not usually found in other inflammatory fevers.

Besides these general symptoms of rigors, febrile movement, and change in the pulse, there are, as a rule, cerebral symptoms. The patient becomes delirious, the degree and intensity of the delirium varying in different cases, sometimes being mild, sometimes acute, like acute mania, and there will be difficulty in confining the patient to bed. Headache is complained of, as a rule, and is often very severe, being referred to the particular part of the head corresponding to the area of inflamed duramater. Not infrequently patients have convulsions, and some of them die in these attacks; in others the convulsions will be repeated from time to time, and finally be succeeded by coma. The coma either succeeds the convulsions or co-exists with them.

Prognosis.—The prognosis is bad. Patients die from the effects of the inflammation or from thrombosis.

Treatment.—The treatment is very unsatisfactory. What we usually do is to apply cold to the head by means of ice-bags, and to make the patient as comfortable and as quiet as possible by the use of bromide of potash, chloral hydrate, and opium. We prefer, as a rule, a mixture of the bromide of potash and chloral hydrate, twenty grains of the former and ten of the latter, to be given every three or four hours, the object being not so much to affect the severity of the inflammation as to keep the patient quiet and comfortable. In some cases the bromide and chloral mixture has no effect, and when this happens we have to resort to the use of an opiate, which should be given in moderate doses. The application of cold to the head is made with a view, partly to act upon the inflammation, partly to diminish the pain or headache, and partly to diminish the delirium. It seems to influence decidedly the delirium and pain, although the inflammation invariably runs its course.

CHRONIC PACHYMENINGITIS.—Chronic pachymeningitis only involves the inner layer of the duramater. It may occur as an idiopathic disease. If it involves the external aspect of the duramater, it may occur as an inflammation secondary to inflammations of the cranial bones. When it occurs as an idiopathic process, and the patient dies, if we have an opportunity of examining the duramater, we find its inner surface covered, not with pus, not with fibrin, but with a layer of organized new tissue. This is composed very largely of cells, with but a scanty supply of basement-substance, and it contains a very large number of new blood-vessels having very thin walls. This layer of new tissue is found most frequently over the convexity of the brain, but it may be so extensive as to line the entire inner surface of the duramater. If, however, it is circumscribed, its favorite situation is that portion of the duramater opposite the convexity of the brain. The blood-vessels which are formed in this layer of new tissue are inclined to rupture, and if they do, we have extravasations of blood into it, which render it so much the thicker. This, however, does not usually take place, but it is an accident which may occur at any time, and as a result there may be a mass of new tissue and blood, which will act as a tumor, and will compress the brain. Sometimes masses are thus formed as large as half an inch, or an inch, or two inches in diameter, which compress the surface of the brain. Less frequently the bleeding from these vessels takes place very rapidly, and the patients die from extensive extravasations of blood compressing the surface of the brain. This occurrence is by no means common.

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Symptoms.—The symptoms of this form of inflammation of the duramater vary with the extent of the inflammatory process, and with the amount of new tissue that is produced. When the inflammation is slight there are no symptoms whatever, and we only discover the lesion at the autopsy. The patient dying from some other disease, we find, upon examination, that the inner surface of the duramater corresponding to the convexity of the brain, either of one side or the other, is covered with a layer of new tissue, showing that an inflammatory process has been going on, and yet during the life of the patient there were no symptoms pointing to it. When the lesion is more extensive there are symptoms which indicate its existence. Dull headache is present a good deal of the time in a considerable number of cases. The pain is sometimes localized, sometimes it is described as a general headache, but it is more commonly characterized as a dull, constant headache; not neuralgic. The intellect is very apt to be impaired, the patients being no longer as intelligent as they were, their memory fails them, and they are not able to attend to their business as well as before. In a certain number of cases the patients no longer walk as well as they did; there seems to be a loss of co-ordination as in locomotor ataxia; they walk very much like patients under the influence of liquor. If they pay attention to it they can walk pretty well, but the moment their attention is withdrawn they walk with a staggering and uncertain gait. Sometimes there will be a loss of consciousness, and this will come on quite suddenly. It will be lost for a few minutes, or a longer time, and then consciousness will return. These are the symptoms we get in the ordinary cases of the disease, in those cases in which the inflammatory process is fairly marked: headache, impaired intellect, unsteady walk, loss of consciousness. In some cases there is another symptom, that of being unable to speak; the patients know well enough what they want to say, but they have difficulty in saying it. They speak slowly and as if they were afraid. They have some difficulty in pronouncing particular words which they want to use.

Progress of the disease.—The course of the disease, as a rule, is slow; it may last for months and for years. Not infrequently its course is intermittent, the patients for a time appearing to be better, or, at all events, they get no worse, the disease seeming to come to a standstill. This may last for weeks, or for months, and then the patients begin to get worse, and the symptoms become more and more marked.

Prognosis.—The terminations of the disease are various. A certain number of the cases go on to become fairly insane; they have chronic insanity, or chronic dementia, properly speaking. Some patients die from the local effects of the inflammatory process, either suddenly from rapid extravasation of blood, or more slowly from pressure of blood and tissue upon the brain. In a certain number of cases the secondary inflammation of the piamater adds to the severity of the symptoms and assist in causing the death of the patient. The larger number of patients, however, die from some intercurrent disease : from pneumonia, pleurisy, or some disease having no connection with the inflammation of the brain.

Treatment.—Concerning the treatment of this condition there is not a great deal to be said. The best that we can do is to keep the patient's general condition as good as we can. If possible, have the patient travel, for we know by experience that in all chronic inflammations a change of climate is one of the most potent agents we have for arresting the inflammation or rendering its course more slow. If a change in climate is impracticable, it is well to have the patient travel from place to place. He should be placed upon the systematic use of iodide of potash and the bichloride of mercury, for both of these drugs seem to have a certain effect upon such chronic inflammation as this. These drugs should be given alternately and in small doses and for a considerable length of time. You may give of the bichloride one-twentieth of a grain three times a day and keep it up for several months, after which its use may be stopped and the patient put upon the iodide of potash, giving five or ten grains two or three times a day, and continue this for several months. It is, of course, very difficult to say what good these remedies do, but still I think it is well to give them, for they do no harm to the patient, and there is, at all events, a possibility that they may be of service in delaying the progress of the inflammation.

LECTURE V.

INFLAMMATIONS OF THE MEMBRANES OF THE BRAIN—CONTINUED.—PACHYMENINGITIS.—CONCLUDED.—MENINGITIS.

Syphilitic Pachymeningitis, Pathology, Symptoms, Diagnosis, Prognosis, and Treatment.—Anatomy and Relationships of the Piamater.—Acute Meningitis; Pathology, Etiology.—Symptoms in Idiopathic Cases; Duration, Prognosis, Treatment.

SYPHILITIC PACHYMENINGITIS.—*Gentlemen:* We began to-day the consideration of syphilitic pachymeningitis, that form of pachymeningitis which occurs in the course of constitutional syphilis.

Pathology.—The inflammatory lesions in this disease are somewhat different from ordinary inflammatory lesions. The product of inflammation is usually circumscribed, and it forms tumors, which are called *syphilitic gumma tumors.* These gumma tumors are composed, at first, principally of small round, oval, and fusiform cells, with a little basement-substance. They have a natural tendency to degenerate and become converted into granular matter. This degenerative process may be either a dry or moist one. If it is dry the gumma tumors become converted into a dry mass, resembling cheese, and this condition is spoken of as *cheesy degeneration.* If the degenerative process is moist, then these tumors are converted into something that looks like abscess, the outer layer forming the wall of the abscess, while the cavity is filled with thin purulent-looking fluid, which, though it looks like pus, does not contain pus-cells, but consists of serum, holding in suspension principally granules and degenerated cells. Sometimes these gumma tumors become the seat of a real suppurative process, and when this is the case, that which was at first a gumma tumor becomes an abscess containing true pus. These tumors are found either upon the external or internal surface of duramater; very frequently in both positions. In some patients they are very apt to be multiple. We often find the same lesions involving the periosteum, which covers the cranial bones. Tumors are found upon the external surface of the cranial bones, and the cranial bones themselves are sometimes involved, and destroyed to a considerable extent by them. These are, then, lesions of syphilitic pachymeningitis; the formation of gumma tumors upon the inner and outer surface of the duramater, in the substance of the cranial bones, and between the periosteum and the bones themselves.

As a complication of the above lesions, we have inflammation of the piamater, and this may be either simple meningitis, or it may be syphilitic meningitis, with the production of fibrin, serum, and pus, or it may be syphilitic meningitis, with the production of gumma tumors in and beneath the piamater.

Symptoms.—Of the symptoms of this condition the most prominent, and the one most to be relied upon, is a persistent headache. Sometimes this will be the only symptom, a persistent headache which does not yield to any of the ordinary remedies. In other cases there are, in addition to headache, other symptoms. Patients may suffer from attacks of unconsciousness which last several minutes. They may have convulsions, and not unfrequently these will be repeated, sometimes at intervals of a few hours, or days, or perhaps, a week. The intellect may be impaired, the patients appearing dull and stupid. If you leave them to themselves they will sit in their chair or lie in their bed all day, without saying or noticing anything. If you speak to them they will answer you, but if you leave them to themselves they will relapse into the same dull, stupid, apathetic condition, in which they take no notice of what is going on about them. In other cases, again, we have delirium, but usually of a mild character. In some instances there will be loss of eyesight. Patients will become blind, and this condition is due not so much to the meningitis as to the affection of the optic nerve by the same poisoning which induces the meningitis. Patients may also lose their hearing, but this symptom, however, seems to depend more directly upon the pachymeningitis, for, upon examination, we may find no lesion of the ears.

If the gumma tumors become of considerable size, and press upon the brain, and especially upon certain regions of the brain, then the patient will have hemiplegia, paralysis of the opposite side of the body from that upon which the lesion is situated. If the lesion be situated upon the right side, then the patient will have loss of power and motion of the left arm and left leg. Sometimes these patients are troubled with vomiting of food. This may be quite frequent and troublesome.

These are the ordinary symptoms of this condition: Headache, a well-marked symptom, sometimes the only one; attacks of unconsciousness, lasting for several minutes; convulsions; change in the intellectual powers of the patient; delirium; sometimes loss of eyesight and hearing, sometimes hemiplegia, sometimes vomiting.

Diagnosis.—The diagnosis is always assisted by our knowledge of the syphilitic history of the patient. If a patient comes suffering from persistent headache, which has lasted for several weeks, and which does not yield to ordinary remedies, and if we obtain a syphilitic history, then we may infer with some probability that there is a pachymeningitis going on. If, in addition to the headache, we get other of the symptoms which I have mentioned, then the diagnosis becomes still more probable. If we find gumma tumors of the cranium situated superficially, so that we can feel them, then the diagnosis is still more certain.

Prognosis.—The prognosis of this condition depends very much upon whether or not patients are properly treated. If the disease is left to itself, the natural termination is the death of the patient. The gumma tumors continue to increase in size, the piamater becomes inflamed, the brain is compressed, and patients die either in an attack of convulsions, or in a comatose condition. If patients can be properly treated, and the disease is not too far advanced before treatment is commenced, then the prognosis is much better, the symptoms will disappear under treatment, and the patient will return to a natural condition, and will be apparently well.

This favorable termination is not always a permanent one. Patients give up the treatment, and pass from under your observation, but in one or two years they may come back to you with the same symptoms which they had when they first presented themselves for treatment, so that eventually the prognosis is bad, though the temporary prognosis is quite good, for you can usually expect that patients will get better at the particular time when you treat them.

Treatment.—The treatment is very simple. It is the ordinary treatment for late syphilis. Mercury or iodide of potassium, either together or successively, are the remedies employed. In the later stages of syphilis iodide of potassium is of more service than mercury alone, hence we use either mercury and iodide of potassium together, or iodide of potassium alone. There is no absolute rule in reference to the dose. You are to give enough to get the desired result, and the amount which it is necessary to give varies very much. In some patients five grains of iodide of potassium will be sufficient to cause the gradual disappearance of the symptoms. In other patients this amount of the drug will be of no use whatever, and it will be necessary to give ten, fifteen, twenty, or thirty grains, or even a drachm three times a day, in order to control the symptoms. You must give a sufficient quantity to get the desired effect, which is the gradual disappearance of the symptoms.

INFLAMMATIONS OF THE PIAMATER.

Anatomy and Relationships of the Piamater.—Now, in regard to the inflammations of the piamater. The piamater is, as you know, the membrane which invests the brain, and which invests it very closely, sending down prolongations into the sulci and fissures of the brain. This piamater is very abundantly supplied with blood-vessels, which communicate with the cortex of the brain. It is not necessary to divide this into two parts, the arachnoid and piamater. For our purpose it is only necessary to speak of it as a single membrane. If it is inflamed at all, its entire thickness is involved in the inflammatory process. It is a connective-tissue membrane, composed of connective-tissue fibres and connective-tissue cells, which are arranged in a somewhat peculiar manner, with which, however, it is not necessary for me to trouble you.

Forms of Piamater Inflammations.—The inflammations of the piamater are acute meningitis, chronic meningitis, tubercular meningitis, and syphilitic meningitis.

ACUTE MENINGITIS.—Acute meningitis, or acute leptomeningitis as it is sometimes called, gives us the following lesions.

λέπτος, thin, ligh

Pathology.—In a certain number of cases that die we simply find the membrane a little thicker than it should be, with its surface no longer of a glistening appearance, but dull and sodden. The blood-vessels contain rather more blood than normal. There is no coagulum, serum, or pus. If we examine it with a microscope, however, we find the reason why the membrane is changed in appearance; there has been a production of new connective-tissue cells in the piamater. This variety of meningitis is very easily overlooked at the autopsy. Persons who are not familiar with it will look at the piamater and say that no meningitis has existed. But this form of meningitis produces well-marked symptoms, and may lead frequently to the death of the patients.

other

In the cases of death from meningitis, we find the ordinary products of inflammation, fibrin, pus, and serum. The relative proportions of these products vary. Sometimes there will be a great deal of pus, and not much fibrin or serum. In other cases there will be a considerable amount of fibrin, and very little pus or serum. Again, the serum will be larger in amount than the other inflammatory products. The whole amount of fibrin, serum, and pus, also varies. In some patients they will be very abundant; in others they will be very scanty.

The products of inflammation are not upon the surface of the piamater, but are infiltrated into it or between the piamater and the surface of the brain. Sometimes there will be so much pus as to give the appearance of little abscesses between the piamater and the surface of the brain. In a considerable number of cases the inflammation will not be confined to the piamater of the brain, but will extend to the piamater which covers the spinal cord. This occurs in a considerable number of cases of meningitis.

Etiology.—Acute meningitis is not very often directly idiopathic, that is, it does not very often occur without some known cause. As an idiopathic inflammation it is more common in children than among adults. It is rare in children but still more so in adults. The conditions under which acute meningitis in adults occurs, are especially injuries of the cranial bones, fractures, or severe blows to the cranial bones, inflammation of the cranial bones, and suppurative inflammation of the middle ear. This latter cause very frequently gives rise to this form of inflammation. The different inflammations of the duramater not infrequently give rise to acute inflammation of the piamater.

Then there are certain diseases in which acute meningitis occurs as a complication, as acute Bright's disease. Pyæmia also gives us a certain number of cases. It is tolerably common in the acute lobar pneumonia of adults. Every winter, I suppose, I see as many as two or three cases of acute meningitis occurring as a complication of acute lobar pneumonia. In the exanthemata, such as scarlet fever and measles, it sometimes occurs as a complication; not very frequently, however. Sometimes it occurs as a complication of typhus and typhoid fever, most frequently as a complication of suppurative inflammation of the middle ear, pneumonia, and typhus fever.

Symptoms of Acute Idiopathic Meningitis.—The symptoms of acute meningitis will vary greatly, according as it is an idiopathic, secondary, or complicating condition. The symptoms of meningitis may be obscured by the symptoms accompanying the cause of the inflammation, or the general disease, from which the patient is suffering.

Let us suppose an uncomplicated case of idiopathic meningitis. In these cases there is a prodromic period, a period of several days, during which the patient presents certain symptoms, but does not consider himself sick. The symptoms which belong to this period are irritability of temper and irregular headache. The headache is sometimes spoken of as quite severe. Others say that it is not very severe, but that they are conscious of a certain amount of discomfort. The patients are also apt to suffer from sleeplessness at night. They also experience a feeling of general indisposition. These are the symptoms belonging to the prodromic period. Sometimes the patient will continue to attend to his business, although feeling sick all the time. After the lapse of a few days the symptoms become more marked, the headache becomes more severe; there is no longer any question about its being a severe headache; then delirium comes on, and in some cases it will come on very rapidly. A patient who has been down town all day, attending to his business, on returning home may, within an hour, become violently delirious. In other patients the delirium will come on slowly, requiring several days for its full development. Some patients even require to be tied in a bed; others manifest a milder form, a wild, muttering delirium, the patient being easily controlled. There are apt to be convulsive movements of the voluntary muscles. Most frequently the muscles of the face are involved. Owing to the convulsive involuntary contraction of the voluntary muscles of the face, the patient looks as if he were making faces at you, as he lies in bed. In other cases the twitching will involve the muscles of the extremities: a patient will suddenly throw up his hand or leg. In other cases the twitching will become more manifest, ending in well-marked general epileptiform convulsions. The patients are apt to become drowsy, the drowsiness alternating with delirium.

When the disease has advanced far enough, and especially if the case is to prove fatal, the patient will become fairly comatose—perfectly unconscious. He will lie in bed with his pupils dilated, and will not answer questions. Congestion of the conjunctiva is apt to occur, which will bring on a conjunctivitis. This occasionally takes place during the prodromic stage, and under such circumstances is of help in making a diagnosis.

When the disease is fairly developed there is usually photophobia. The patient dreads the light, and wants to have the room dark all the time. This seems to be due, in some cases, to the conjunctivitis. In other cases conjunctivitis occurs independently. We also get internal strabismus by the contraction of the internal rectus. There is, very generally, hyperæsthesia of the skin, the patient complaining of pain upon slight pressure, or removal of pressure, or when being moved. The hearing is sometimes unnaturally acute, and the patients complain of noises in the room. Any sudden noise will make them start up.

This unnatural sensitiveness is succeeded by the opposite condition. The patients no longer hear as well, and they sometimes appear to be almost entirely deaf. If the disease extends to the membranes of the spinal cord, then there may be opisthotonos, that is, contraction of the muscles of the back of the neck, and along the spinal column. The contraction of these muscles will cause the patient to keep his head held back, and to lie upon his side in bed with his body bent backward. Accompanying this there will be a certain amount of pain and tenderness along the vertebral columns. There is always a febrile movement. This is a general symptom accompanying a local inflammation. The febrile movement, however, is irregular, that is, the temperature does not follow any regular course, varying in many cases from 100° to 104°; in many cases not above 102.° It is a well-marked febrile temperature, but not excessively high. The rise and fall of the temperature in these cases is no indication of the progress of the disease. It may be lower when the patient is doing badly, or higher when the patient is doing well. The pulse at first is usually frequent, but when the disease is fairly developed it will be 80, 70, 60, or even 40 per minute. Then, if the case goes on to a fatal termination, the pulse is at first full and rapid, then full and slower, and finally rapid and feeble.

At the commencement of the disease the tongue has usually a thick, white, furry coat, which remains moist. After the disease has lasted for several days, however, and especially in fatal cases, toward the end of the disease, it becomes dry and of a brown appearance, looking like the tongue of a patient suffering from typhoid or typhus fever. In a certain number of cases there is a disturbance of the stomach; there is vomiting; this may mark the onset of the disease, occurring during the first twenty-four hours, or forty-eight hours, and then cease altogether; or continue at intervals during the continuance of the disease. The bowels are as a rule, constipated, that is, they do not move without the action of medicine. This rule is not in-variable, and especially is this the case at the close of the disease, where we have, in some instances, a little diarrhœa. These, then, are the symptoms of acute meningitis, when the disease occurs idiopathically, and is not obscured by other symptoms. The change in the pulse, the condition of the tongue, the derangement of the stomach, and the state of the bowels are all general symptoms accompanying a local inflammation. In fact, the whole history of the disease is that of a local inflammation, accompanied by constitutional symptoms.

Duration.—The duration of acute meningitis varies from thirty-six hours to four weeks, the average duration being about two weeks. Many cases which you see will run their course in a week, especially fatal ones. Patients who are to recover are sick for a longer time before they become thoroughly convalescent.

Prognosis.—The prognosis is, as a general thing, bad. The larger number of patients die. But on the other hand the prognosis is not hopeless. It is always possible that a patient may get well, and a certain number do, but the prognosis is better in children than in adults.

Treatment.—The treatment is directed partly to subdue the local inflammation, and partly to allay the severity of the constitutional symptoms. To subdue the local inflammation, the most powerful agents are, local bloodletting and the application of cold. If the patient is a strong and vigorous adult, or, even if it is a strong and vigorous child, you may take blood, either from the temples and nape of the neck, by leeches, or by wet cups. The time to do this, however, is at the commencement of the disease. If you see the patient within the first forty-eight hours of the attack, and he is tolerably strong and robust, then I think local bloodletting is of service. But, if you do not see the patient till after this time, especially if it be several days after the beginning of the disease, then local bloodletting can no longer be of any service to the patient. The inflammation is no longer going on in the same way, the products of inflammation are already formed, and the patient is suffering from the presence of these more than from the inflammation itself. So that time is an important feature in taking blood.

Cold is applied to the head, usually best by means of ice-bags, and, in order to apply it thoroughly, it is necessary to cut the patient's hair closely, if the patient has a pretty thick head of hair, and especially if it be the long hair of a woman. It is necessary to cut it, in order to make a thorough application of cold. Cold should be applied to as much of the external surface of the cranium as possible. An ice-bag may be placed at the nape of the neck, and one or more ice-bags so placed as to completely cover in the cranium. If you apply cold you should apply it thoroughly. The application of cold, like bloodletting, is of especial service during the first two or three days of the disease, but you may use it with advantage for four, five, or six days, as the application of cold not only has the effect of diminishing the severity of the inflammation, but it makes the patient feel more comfortable. It relieves the pain, headache, and general irritability, from which these patients suffer, and sometimes has the effect of lessening the delirium.

These are the two external agents which are of the greatest service in diminishing the severity of the inflammatory process. Besides these, we may use drugs. The drugs which have been most recommended for their effect upon the inflammatory process are, iodide of potassium and ergot. Both these drugs are given frequently as a matter of routine practice.

LECTURE VI.

INFLAMMATIONS OF THE MEMBRANES OF THE BRAIN—CONTINUED.—MENINGITIS.

Acute Meningitis Secondary to Chronic Suppurative Inflammation of the Middle Ear.—Acute Meningitis Complicating Pneumonia.—Chronic Meningitis; Post-mortem Appearances, Symptoms, Prognosis, and Treatment.—Tubercular Meningitis.—Occurrence in Children.

ACUTE MENINGITIS.—*Gentlemen:* We were speaking yesterday of acute meningitis, and we considered those cases in which the inflammation occurs as an idiopathic disease with well-marked symptoms—symptoms not obscured by those of other diseases. In these idiopathic cases we found that the disease not infrequently was preceded by a period of prodroma; that when the disease was fairly developed we had well-marked cerebral symptoms, and well-marked constitutional symptoms. The disease runs its course within a period varying from thirty-six hours to four weeks. When we have this same inflammation occurring as a secondary condition, or as a complicating condition, the symptoms are not apt to be as well marked, the diagnosis is more difficult, and the cases are more apt to be overlooked.

Acute Meningitis, Secondary to Chronic Suppurating Inflammation of the Middle Ear.—In the first place, let us suppose it to occur as a secondary condition and in its most frequent form, as a secondary condition to suppurative inflammation of the middle ear. In these cases the suppurative inflammation of the ear may be either acute or chronic. If it is acute the history of the patient will be something as follows: Some three or four weeks before you see the case the patient will have suffered from a severe cold, which was followed by pain in one ear, and this pain may have been accompanied by rigors and a febrile movement. The febrile movement may be intermittent, and I have known such cases to be mistaken for cases of intermittent fever on account of the intermissions between the periods of febrile movement. After patients have gone on this way for three or four weeks, having more or less pain, with severe headaches or fever, they will become worse, but not in a very definite way. The manner in which they become worse will be by a persistence of the fever. It will be continuous, and the patient may become delirious and be much sicker afternoons than in the earlier part of the day, and confined to his bed. In this delirious condition, with irregular but continued febrile movement, with a pulse which becomes slower after the disease is fairly developed, these patients will continue for several days, then the pulse will become more rapid and more feeble, and the delirium will be succeeded by drowsiness and coma, and the patient will die.

At the autopsy you will find that inflammation of the middle ear has been going on all the time. There will be a layer of fibrin and pus, and you will find a thrombosis in the venous sinuses of the duramater. When you look at the piamater you will find it infiltrated with fibrin, pus, and serum, or simply with new connective-tissue cells. Here the symptoms are not as well marked as in cases of idiopathic meningitis. It is a good general rule whenever a patient becomes seriously ill and you know that there is suppurative inflammation of the middle ear, to always think of the possibility of the existence of meningitis.

We will suppose, on the other hand, that a patient has been suffering from chronic suppurative inflammation of the middle ear. This condition lasts for months and years, sometimes a little better, sometimes a little worse, but there being at all times a discharge from the ear. At some particular time the patient will have a febrile movement with or without rigors. This will not run very high, but it will be marked and the patients will be sick enough to be confined in bed, and will be somewhat delirious. They will complain a good deal of headache, and will very likely refer it to the side of the head upon which the diseased area is located. They may have hyperæsthesia of the whole body. They may become maniacal, and delirium will be followed by stupor, coma, and death. At the autopsy you will find the same as before.

Meningitis Complicating Pneumonia.—We will now suppose that meningitis occurs as a complication of pneumonia. The patient will be attacked with the ordinary symptoms of pneumonia; with rigors rapidly succeeded by a febrile movement, the fever running up to 103°, 104°, or 105° on the second day of the disease, there being pain in the side, with a characteristic cough, and characteristic sputa. The patient will continue in this condition for two or three days, but you will usually notice that he is more restless than in an ordinary case of pneumonia. We will suppose that, at the end of the first week, the restlessness and irritability of the patient have become excessively marked. He does not sleep at all, either day or night, except for a short time; he complains of bad dreams, of seeing objects which do not exist in the room, and he will complain of headache. He will then become fairly delirious, and you will now observe convulsive twitchings of the muscles of the face. These are apt to be quite marked. The patient will become stupid, then comatose, and die in the same way as the other cases. When the case is secondary to pneumonia, you have to remember that in pneumonia there are cerebral symptoms of much the same character as those in meningitis. There are a considerable number of cases of pneumonia in which the patients are very restless, very irritable, delirious, and die comatose, and in which there will be no meningitis whatever. When you come to see these patients you will appreciate that there is a difference between those having pneumonia with cerebral symptoms, and those having pneumonia complicated by meningitis. The distinction can only be fairly appreciated by actually seeing the patients and observing their condition in the two instances.

The complicating and secondary cases of meningitis usually run rather a short course, the patient dying within a week after the development or beginning of the disease. They may not always die from the meningitis, but they are pretty sure to die from one or other of the two diseases. Complicating meningitis in the majority of cases is fatal.

The treatment is the same as in the idiopathic form of meningitis. The great difficulty in these cases is to make out the diagnosis, to determine the fact that meningitis has been developed.

CHRONIC MENINGITIS.—*Post-mortem Appearances.*—In chronic meningitis we have the inflammation involving the whole of the piamater, involving the piamater over the entire surface of the brain, or it may be confined to the portion covering the base of the brain, or to that which covers the convexity of the brain. The change which we find taking place in the piamater is that it is thickened and more opaque than it should be; it is denser and firmer than normal. There is an infiltration of the tissue of the piamater with serum, fibrin, pus, and with new connective-tissue cells: the pus is not usually very abundant. The degrees to which these lesions are developed vary very much in the different cases; the piamater may be a good deal thickened—it may be one-eighth of an inch thick, or more, or so slight as to be hardly measurable. At times the inflammatory products are present in large amounts, again they are scanty. In the well-marked cases there are usually pronounced changes in the superficial portions of the cortex of the brain; these may become infiltrated with cells, softened, hardened, or sclerosed. Not infrequently we find organized adhesions between the external surface of the piamater and the internal surface of the duramater, and between the piamater and the brain. Quite frequently there are changes in the ventricles of the brain; they may contain an increased amount of serum which may be either clear or turbid.

These lesions must not be confounded with the degenerated condition of the piamater with which we meet from time to time. In a moderately large number of autopsies it is found that there is considerable thickening of the piamater. This is not inflammatory thickening, but is due to a degenerative process, there being no pus, and only a moderate increase of connective-tissue cells. This degenerated condition of the piamater is found in connection with many diseases; it is found in old cases of Bright's disease, cirrhosis of the liver, and old alcoholic cases. This condition is not what I mean when I speak of chronic meningitis.

Occurrence.—Chronic meningitis is a disease of adult life. It is possible for it to occur in children, but the very great majority of cases occur in adults. When it occurs as an idiopathic disease, it usually occurs in poor people—in people who have not had enough to eat, and have had too much to drink. Then we have it occurring secondarily in fractures and inflammations of the cranial bones, and as a complication of chronic Bright's disease; also as a direct result of chronic alcoholic poisoning.

Symptoms.—The symptoms of chronic meningitis are very much like the symptoms of chronic pachymeningitis which we have already described. As in chronic pachymeningitis, so in chronic meningitis, the degree to which symptoms are developed varies in different cases. There are patients who can be said hardly to have any symptoms; they die of some other disease, and at the autopsy the existence of a moderate degree of chronic meningitis is demonstrated, and this has not produced any symptoms during life. In other cases there are cerebral symptoms. Persistent headache, worse at some times than at others, but rarely absent for any length of time, is one of these. The mental faculties are changed; the patients become dull, stupid, and lose their memories; are unable to attend to their business, and their temper is likely to be changed, the patients becoming unnaturally peevish and irritable. Disturbance of the mental faculties may become very marked indeed, so that the patient will be insane. Occasionally patients have delusions, and, as in insanity, they imagine they are persecuted by foes, and that all sorts of bad things will happen to them or their families. The irritability is apt to be accompanied by sleeplessness at night, and yet in the daytime these patients are in a drowsy condition. They may become fairly delirious. The delirium is not apt to be active, but of a mild character. If you speak to patients in this condition they will answer you rationally upon some subjects, and then if you leave them to themselves they will talk at random. There may be disturbances of sensation—peculiar feelings in different parts of the body. Some of the patients will tell you that one of their legs is always numb, or that they have pains in their arms, or legs, or that there is a hyperæsthesia of some parts of the skin. There are cases in which there is a loss of motion in some of the muscles in addition to the above symptoms. The patients may have attacks of unconsciousness, or there may be times when they will fall to the ground without being unconscious. They will be sitting in a chair or standing, and suddenly fall to the ground, and yet retain their presence of mind and be able to answer questions. They will fall without being unconscious, and will not be able to give any reason for so doing. At other times they will be fairly unconscious for a variable length of time. Sometimes these patients will have epileptiform convulsions, and they may have these at intervals of months. There may be disturbances of the stomach, the patient being troubled with nausea and vomiting. The bowels are usually constipated. As a rule there is no febrile movement. Occasionally, these irregular symptoms last for months, sometimes for years, the condition of the patient gradually getting worse. There is considerable difference in the cases as to the peculiar way in which the symptoms are developed. The inflammation itself is a very chronic one, and in a certain number of cases the symptoms will be developed very slowly indeed, so gradual that the patient or his friends can hardly tell you when he began to be sick. But there are other cases in which the onset of the symptoms is acute. The patient, so far as known to himself or friends, will have been apparently healthy. On some particular day he will have an attack of convulsions, or suddenly become unconscious or delirious, and then, after this, other symptoms may make their appearance. It is very difficult to understand this apparent sudden invasion of the disease, but you will find this to occur in a certain number of patients.

Prognosis.—The prognosis of this condition is not good. The disease is usually of considerable duration, but the patients as a rule do not get well; some of them go on and become fairly insane; they become permanently insane and pass from the category of sick persons to the category of insane persons. In other cases the patients seem to die from exhaustion; they lose their appetite, they gradually lose flesh and strength, and finally die emaciated; you can hardly say why they die, only they die; you find no particular cause of death. Other patients die with cerebral symptoms; they die comatose. Still other patients die from intercurrent diseases; from pneumonia, pleurisy, or some disease which is developed, and carries the patient off.

Treatment.—The treatment is not at all satisfactory. The drug which seems to be of as much service as any other is iodide of potash. Under the use of this patients will sometimes seem to improve, and even for a considerable length of time; but in these cases, if the improvement is tolerably permanent, there is some doubt if the case is not really one of syphilitic meningitis. As a certain number of patients do improve under the use of iodide of potash, I do not know of any better treatment than this, at the same time attending to the general nutrition of the patient as well as you can.

TUBERCULAR MENINGITIS.—This form of inflammation of the pia-mater occurs both in adults and in children. It is more common, perhaps, in children than in adults, and both the lesions and symptoms are not exactly the same in adults as in children.

Occurrence in Children.—We will consider first the disease as it occurs in children. If a child dies of tubercular meningitis, or acute hydrocephalus, as it is very often called by English writers, and we have the opportunity of examining the head, we find the duramater usually unchanged. In exceptional cases we find miliary tubercles on its inner surface. When we have removed the duramater and looked at the surface of the brain, we find that the brain appears too large for the skull, the convolutions of the brain look flattened and compressed. If we look at the piamater we find, in a certain number of cases, that it is congested, and, in a certain number of other cases we find this congestion absent. In still other cases we find the piamater infiltrated with fibrin, serum, or pus, and whether it is congested, pale, or infiltrated with fibrin, serum, or pus, we find imbedded in it small, whitish, grayish bodies, called miliary tubercles. These lesions may be confined to the base of the brain, or over the whole of the surface of the brain. Tubercles may be in small numbers or may be in great numbers. They may be so small that you can scarcely see them with the naked eye, or much larger. They are always more abundant in the sulci and fissures of the brain than in that part of the piamater which covers the convolutions. If we look at these miliary tubercles with a microscope we find they consist of accumulations of round cells about the blood-vessels. The blood-vessels of the piamater are naturally surrounded by what is called a perivascular sheath, which forms a covering to them. In a certain number of cases we find that the tubercles form localized swellings of these perivascular sheaths. Besides this circumscribed inflammation, you may discover with it a more diffuse, or less marked inflammation, and there will be some cells between the perivascular sheaths and the walls of the blood-vessels. In other cases the miliary tubercles have a different structure; they are formed of larger cells, of an accumulation of polygonal cells. In these cases you will find single cells in other parts of the piamater forming a more diffuse inflammation. This is the structure of the bodies which we call miliary tubercles; they are sometimes merely an accumulation of round cells in the perivascular sheaths, sometimes an accumulation of polygonal cells.

When we cut the brain open we find that the ventricles are larger than they should be, and are distended with serum. The ependyma are sometimes changed—thickened or softened. This distention of the ventricles with serum is a regular part of the lesion in children, but we will see presently that it does not obtain in the case of adults. This is one of the practical differences between the tubercular meningitis of children and adults. The lesions both of the piamater and ventricles are quite apt to extend down the spinal cord. When this takes place the piamater of the spinal cord will present the same changes as that of the brain, and the central canal of the spinal cord will be dilated in the same way as the ventricles of the brain. In most of the patients who die of this disease the tubercular process is not confined to the brain and spinal cord, but we find miliary tubercles in other parts of the body. It has always been a disputed point whether we ever have tubercular meningitis without the presence of miliary tubercles in other parts of the body, and this question is still answered in different ways by different authors. There can be no doubt but that, in the majority of instances, tubercles are found in other parts of the body. I think, however, it is well to admit the possibility of the development of tubercles in the piamater independently of their appearance in other parts of the body. The majority of cases of tubercular meningitis in children are in those under five years of age. There are annually reported a number of cases of death from this disease under one year of age, but such statements are always, I think, to be received with doubt. If patients of this age have tubercular meningitis they do not present well-marked symptoms, so that unless you know the diagnosis has been confirmed by an examination after death, you cannot place much reliance upon the case being one of tubercular meningitis. Children in whom this disease occurs are, as a rule, those whose parents or relatives have suffered from pulmonary phthisis.

LECTURE VII.

INFLAMMATIONS OF THE MEMBRANES OF THE BRAIN—CONTINUED.—TUBERCULAR MENINGITIS.

Tubercular Meningitis in Children.—Pathology, and Symptoms in First, Second, and Third Stages; Prognosis and Treatment.—Tubercular Meningitis in Adults; Pathology, Symptoms, Prognosis.

TUBERCULAR MENINGITIS IN CHILDREN.—*Gentlemen:* We were speaking at the close of our last lecture of the occurrence of tubercular meningitis in children.

Pathology.—After death, in this disease, we find in the piamater, and over the surface of the brain, minute bodies called miliary tubercles. In addition to these we may or we may not find other products, as fibrin, serum, pus, and new connective-tissue cells: the tubercles are constant, the other products are sometimes present, sometimes absent. These changes may be confined to the piamater of the brain, or involve the piamater of the spinal cord, or still less frequently, the inner surface of the duramater. Superadded to these changes in the piamater, we find changes regularly taking place in the ventricles of the brain, they being distended with serum and having their walls thickened. On the other hand, occasionally, the walls of the ventricles are softened, and this process may extend to the adjacent brain-tissue.

The disease is most frequent in children under five years of age, and in those whose relatives are subject to chronic pulmonary phthisis.

Symptoms in Children.—In describing the symptoms of tubercular meningitis in children it is customary to divide the course of the disease into three stages, and to speak of the symptoms which belong to each stage. This division, however, is merely for convenience, and you must not expect that you will by any means always find these stages well defined: the stages will run into each other, or one or more may be absent.

First Stage.—In the first stage the symptoms are not very well marked. The child becomes somewhat peevish and irritable; the stomach is disturbed, as shown by loss of appetite and by vomiting from time to time; or the child may complain of a feeling of nauseousness, and of discomfort in the region of the stomach, and its tongue will be coated. Apparently these patients suffer from headache, for while playing they will sometimes put their hands to their heads, as if they experienced pain there, but it is always more or less difficult to determine such points, as they are not usually old enough to tell you what they feel. These children are easily tired out, and want to lie down more of the time than they should, or want to be carried about; they do not sleep well at night, and yet they may sleep more in the daytime than when they are well. The bowels are most often constipated. At times, when asleep, they will grind their teeth, but this symptom is not peculiar to these children.

There will be times during the day when the child will appear as well as ever, after which it will manifest the disposition of which I have spoken. Neither the temperature nor the pulse is peculiar. These are the symptoms of the first stage, and, you observe, none of them can be considered characteristic of any disease. The first stage commonly lasts from two to five days; it is possible for it to last a little longer or be absent altogether, or only last for so few hours that it passes unknown.

Second Stage.—When we come to the second stage of the disease it is at once evident that the child is suffering from some serious illness. The child no longer feels well enough to be up and about, but sometimes is still well enough to be dressed, though showing very little disposition to move about and play. The expression of the child's face is apt to be changed to an anxious one, and not infrequently its cheeks will be more flushed than normal, which condition will come and go. There is photophobia, the child turning its head so as to avoid the light, and closes its eyes if there is very much light in the room. Sometimes one pupil will be dilated and the other contracted, and quite frequently there is external strabismus of one eye. The child is unnaturally drowsy, and lies quietly a good deal of the time.

This drowsiness alternates with periods of excitement, during which the child seems to be suffering pain; it will utter sharp, piercing cries, or simply moan, or complain for a number of minutes, sometimes for half an hour, and then this period of excitement will pass off, the child lapsing into a drowsy condition, in which it takes no material notice of what is going on about it. The length of the periods of excitement, compared with those of drowsiness, varies in different cases. In some, the restlessness and periods of apparent suffering will continue during a good deal of the time. These are the most distressing cases to take care of, for they are constantly experiencing bodily pain. Then there are children in whom these symptoms of restlessness and irritability are almost wanting, or in whom they exist for so short a time that little attention is paid to them. Such children will lie quietly upon the bed; they can be aroused, and will say a few words, then go off into this quiet, pathetic condition, as if sleeping. These children are sometimes so very quiet that you may think there is nothing the matter with them. You may go into the room and find the child lying upon the bed apparently breathing as in natural sleep; but this sleep continues and the child does not arouse. Between these extremes of a great deal of restlessness and a great deal of stupor there are all grades as to the amount of restlessness and stupor which exists.

The disturbances of the stomach, which were present during the first stage, may continue in the second; the child may from time to time vomit. Constipation of the bowels also continues, no movements taking place except by the use of enemata, or by the use of medicine. The pulse, during the first part of the second stage, is rapid and full; then, as the second stage becomes more fully developed, the pulse will become slow, and it is very apt to become irregular. A beat will be lost from time to time, or the pulse will be rapid during some parts of a minute, then slow and irregular. This condition of the pulse obtains when the second stage is fairly developed. The breathing becomes less and less forcible, slower and slower, until the child has apparently almost stopped breathing, then it will take a long inspiration, and so this irregular breathing will go on from day to day. There is also a febrile movement, but this is of a very irregular character, there being times when there are remissions in the morning, and again in the evening. The temperature varies during the twenty-four hours, but not with any regularity. Through a considerable part of the second stage of the disease the temperature will not be high; between 99° and 100°; but toward the close of the second stage it usually runs up to 102°, 103°, or 104°.

The function of the kidneys is usually disturbed; the patient passes less urine than normal, and it may contain albumen. Sometimes the urine is suppressed altogether; the patient passes no urine at all for twenty-four, thirty-six, or forty-eight hours; more frequently it is scanty, than suppressed for so long a time. These are the symptoms which belong to the second stage of the disease.

Third Stage.—As we pass from the second stage to the third stage of the disease the child's condition becomes worse. The drowsiness increases into almost complete stupor, the active cerebral symptoms become less and less marked, and the stupor becomes more and more persistent. We may now have convulsions, and these are more apt to affect one side of the body than the whole of the body; there will be convulsive movements of the leg and arm of one side of the body, and none at all upon the other side. These convulsive movements may be followed by loss of power in the arm and leg in which they occurred.

Besides these regular convulsive movements, we have what are called *automatic movements* of the arm and leg; in other words, the child will move one arm or leg, up and down, up and down, for a long time, almost with the regularity of the ticking of a clock; it will go on doing this for hours. If you take hold of the arm you may stop it: but when you let go it will continue to move, up and down. These movements may take place in an arm or a leg, or in both at the same time. Instead of movements of an arm or leg, the automatic movements may be confined to the fingers, and when this is the case the patients usually take hold of something, or pick it. They are especially liable to pick the nose or lips, and to go on picking until they have completely removed the skin, leaving a raw surface; they do not seem to have pain.

There is now no longer any photophobia. The child takes no notice whatever of light. The pupils do not contract, but become fully dilated. The children not only do not fear light, but take no notice of it. You may hold the eyelids open, and bring a lighted candle close to the eye, but they will not take the slightest notice of it. Whether they are really blind we are unable to tell, but the stupor is so great that they take no notice of anything. There is often a peculiar oscillation of the eyeballs—a rolling from side to side. This seems to be an automatic movement of the muscles of the eye, similar to the automatic movement of the muscles of the leg, arm, or fingers, there being apparently no volition on the part of the patient; with this there may be external strabismus.

The temperature is now persistently higher than during the second stage; 102°, 103°, 104°, or 105°, for a greater part of the time. The pulse becomes more rapid, and at the same time more feeble. It may become so rapid that you can hardly count it. If the lesion extends to the spinal cord there may be opisthotonos; the child's head and body are bent backward. The third stage may end by the death of the child in an attack of convulsions. If, however, this is not the case, the child becomes much emaciated. During the second stage of the disease the emaciation is not particularly noticeable, but after the third stage has lasted for several days, then the children begin to emaciate, and the loss of flesh is very marked indeed. The urine may be suppressed altogether toward the end of the third stage; the skin becomes cold; there is what is called a clammy perspiration; the breathing is very much accelerated, and the pulse becomes exceedingly feeble and rapid. The patients become cyanotic; the skin which was first red, then pale, now changes to a bluish livid color, and so the disease advances until the child dies of exhaustion.

During the progress of the disease, in the first and second stages, and in the early part of the third stage, periods of improvement are not infrequent. There will be hours, or parts of days, when the child is much better. It may rouse up, say a few words; it may have a better appearance; the breathing will improve and be more regular, and the child look as if it was decidedly better. This apparent improvement only lasts for a few hours, and then all the symptoms come back again. It is important for you to remember this in the treatment of these cases, for otherwise you may be allured into the mistake of telling the parents that the child is really better. There are probably no cases in which these periods of improvement amount to much, and you must not be misled by them.

The duration of the disease, including the first, second, and third stages, is from five days to three weeks, the average duration being about twenty days. Some patients die earlier, and if so, they are likely to die in an attack of convulsions.

Prognosis.—The prognosis is exceedingly bad. I think there is no reason for believing that children ever get well of tubercular meningitis. We must remember that the symptoms of this disease resemble those of acute meningitis, and that although the prognosis of acute meningitis is bad, children may get well of it; so that the prognosis of a case which is apparently one of tubercular meningitis is rendered doubtful by the uncertainty of the diagnosis. If you could be absolutely certain of your diagnosis, you could be pretty sure of your prognosis, that the child would die; but this certainty is not possible in the greater number of cases.

Treatment.—The treatment of the disease is for the most part the treatment of symptoms. If the urine is scanty, or suppressed during the second stage, one of the best ways of increasing its secretion is to place the child in a hot-water bath for five minutes, then take it out and wrap it in blankets without stopping to wipe it off, and let it remain there for one, two, or three hours. In this way you induce a free perspiration, and besides this the kidneys will often act while the child is thus wrapped. Diuretics are of little service to relieve the restlessness or irritability and apparent pain; the remedies which have been found most serviceable are the bromide of potash and chloral hydrate; one to five grains of the bromide, and from one to three of the chloral, made into a solution, may be given every two or three hours, according to the age of the child. In addition to this, iodide of potash may be given in doses of two to four grains; this seems to act as a diuretic sometimes. If you choose to be still more energetic in your treatment, you can apply a blister to the nape of the neck or cold to the scalp. You can, if you wish, give the fluid extract of ergot in teaspoonful doses.

The energy of your treatment will depend upon the number of cases of tubercular meningitis which you see. The first case you will probably treat pretty thoroughly, and the case will die. The next case you will perhaps do less to combat the disease and more to make your patient comfortable, and the child will die as before, and so you will go on, becoming more and more convinced of the inefficacy of any plan of treatment against the disease, and you will content yourself with simple measures, disturbing the child as little as possible, and making it as comfortable as you can.

TUBERCULAR MENINGITIS IN ADULTS.—*Pathology.*—We come now to tubercular meningitis in adults, and here we find, as before, miliary tubercles in the piamater of the brain. They are most abundant in the fissures, or sulci, of the brain. We also sometimes have in adults large cheesy masses situated in the piamater, and dipping down into the sulci and between the convolutions. These vary in size—they may be as small as a pea or as large as a pigeon's egg. There may, or may not be, other products of inflammation. There may be fibrin, serum, pus, and new connective-tissue cells in addition to the tubercles, or these products may be almost entirely wanting, and even the miliary tubercles very scanty. Tubercles are very apt to be present in parts of the body other than the piamater, especially in the lungs ; but there are cases in which this lesion is confined to the brain. In adults, as in children, the lesion may extend from the piamater of the brain to the piamater of the spinal cord.

Symptoms in Adults.—The symptoms vary with the particular way in which the disease occurs. There are some cases of tubercular inflammation in adults in which it is impossible to make a diagnosis. This obtains in cases where patients recover from what is called general miliary tuberculosis. Sometimes patients have symptoms of a continued fever running along for several days, and then die. Upon examination after death miliary tubercles are found in all parts of their body. There are cases of acute miliary tuberculosis which give no marked cerebral symptoms, so that the diagnosis cannot be made out. Then there are other cases of acute general tuberculosis where we have marked cerebral symptoms. There are also cases of pulmonary phthisis which have cerebral symptoms.

In those cases where we do have cerebral symptoms in adults they are not as well marked as in children. The disease does not run through any such regular succession of stages. There is a feeling of general malaise ; there may be a chilliness or an actual rigor at the beginning of the inflammatory process. Some patients never have a temperature above 101° during the course of the disease ; there are others who have a temperature running up to 106° or 107°. You will find it stated in some of your text-books that the characteristic temperature of tubercular meningitis is high ; in other books it will be stated as low ; both of these statements are perfectly correct. You can hardly say there is any characteristic temperature of this disease in adults.

The stomach is disturbed, the patients lose their appetite, they may vomit from time to time, the tongue becomes coated, and toward the close of the disease it may be brown and dry. There are cerebral symptoms, the patients complaining of headache—some a great deal, others very little. There is apt to be delirium of a mild character at first, and somewhat later on a more active delirium. With this there is likely to be a change in the mental faculties; patients do not understand what you say, and are not able to answer questions rationally. Contractions of the voluntary muscles may take place, especially those of the face, and these contractions may go on to general epileptiform convulsions. The convulsions may be succeeded by paralysis of the muscles which have undergone involuntary movements. There may be hyperæsthesia of the skin, and finally the patient dies either in convulsions, or comatose.

This is the ordinary course of the disease, and its duration is about fourteen days. It is usually an acute process terminating in the death of the patient in about two weeks. There are those who die within a week. Besides these regular cases of tubercular meningitis, there are irregular cases in which the disease runs a more chronic course, lasting for weeks with moderate cerebral symptoms. Then, there will be an increase in the severity of the symptoms, and the patient will die. The acute course is the more common of the two.

Prognosis.—The prognosis is as bad as it is in children, and it is rendered worse by the fact that, in most of the cases, there is not only local tuberculosis of the piamater, but general tuberculosis, or the patient may be suffering from chronic pulmonary phthisis.

Treatment.—The treatment amounts to nothing at all. There is nothing to be done except to keep the patient comfortable.

You will observe the difference between tubercular meningitis in adults and children. In children it occurs as a disease by itself, and the brain-symptoms are so much more marked, that we are in the habit of thinking of these cases not as cases of general tuberculosis, but as cases of tuberculosis of the piamater. It is just the reverse of this in adults. In children the disease runs a tolerably typical course, and is not to be confounded with anything except ordinary acute simple meningitis. In adults the course of the disease is less characteristic, and there are some cases in which it is impossible to make a diagnosis.

LECTURE VIII.

INFLAMMATIONS OF THE MEMBRANES OF THE BRAIN — CONCLUDED. — SYPHILITIC MENINGITIS, THROMBOSIS, AND EMBOLISM.

Syphilitic Meningitis; Pathology, Symptoms, Prognosis, Treatment.—Of Thrombosis in General; Causation, Results of.—Of Embolism in General; Causation, Results of.—Hemorrhagic Infarction; theories.—White Infarction.—Emboli Producing Local Irritation.

SYPHILITIC MENINGITIS.—*Pathology.—Gentlemen:* The last form of meningitis which we will speak of is *syphilitic meningitis.* In syphilitic inflammation of the piamater the lesions are of much the same character as we find in syphilitic inflammation of the duramater; that is, we find gumma tumors. The inflammation usually involves circumscribed portions of the piamater. These tumors are especially apt to be formed where the piamater dips down between the convolutions of the brain into the sulci. Some of these are quite small, some as large as a pigeon's egg, and some are even as large as a hen's egg. In these large gumma tumors the process is not confined to the piamater, but extends to the brain-substance, portions of it being involved and occupied by them. In a certain number of cases, in addition to the formation of these tumors, there will be a general inflammation of the piamater of an ordinary character, with the production of fibrin, serum, pus, and new connective-tissue cells.

Symptoms.—The symptoms will vary with the size of the gumma tumors, and with the existence or non-existence of general inflammation of the piamater, together with the rapidity with which the inflammation is developed. If there is considerable inflammation of the piamater, besides the presence of the gumma tumors, and the process is acute, the patient will have symptoms resembling, and practically the same as ordinary acute meningitis. If there exists general inflammation with gumma tumors, and the process develops slowly, then the patient will have symptoms identical with simple chronic meningitis. If the tumors reach a considerable size, and there is no general inflammation, then the symptoms are those of tumor of the brain. We have, then, these three sets of cases, in all of which there is a syphilitic lesion, the symptoms varying as I have described.

Treatment.—The treatment of this condition is the same as the treatment of syphilitic pachymeningitis; that is, the treatment of constitutional syphilis, by means of mercury and iodide of potassium.

Prognosis.—The prognosis is the same as that of syphilitic pachymeningitis, except that it is not quite as good. If the piamater is involved in a syphilitic inflammatory process the symptoms are marked, and the danger to the life of the patient is greater than in syphilitic pachymeningitis.

THROMBOSIS AND EMBOLISM.

The next morbid lesion of the brain which we will consider is that condition which is produced by accumulations within, and stoppage of the cerebral blood-vessels. This is effected either by means of what are called *thrombi*, or what are called *emboli*. Before going on with the description of these conditions as they occur in the brain, we will stop to consider the processes of thrombosis and embolism in general.

OF THROMBOSIS IN GENERAL.—A thrombus is a clot formed during the life of the patient, either in the cavities of the heart, in the arteries, in the veins, or in the capillaries. Such a clot is composed principally of fibrin, and entangled in it are a certain number of white and red blood-globules. A thrombus is at first nothing but an accumulation of blood, and is just such a clot as you can have formed outside of the body, by bleeding a patient and collecting the blood in a vessel. The peculiarity of this is, that it takes place during the life of the patient and in situations where blood ought not to accumulate, namely, in the cavities of the heart, in the arteries, in the veins, and in the capillaries.

When blood accumulates in this way it may either completely fill and occlude the particular vessel in which it accumulates, or it may only partly fill it. The accumulation of blood in the aorta may entirely obstruct it, or only partially do so. Or, if the thrombus be in a large vein, it may fill and distend it, or there may be only a small thrombus partly filling the vein.

These thrombi, if seen soon after their formation, look like ordinary blood-clots, or coagulum in the vessels. If you draw off blood from an animal and put it in a glass, the clot will be at first red, and this is what we see in a certain number of thrombi occurring during the life of the patient; others are not red, but white. You can obtain this white clot outside of the body by agitating the blood in the vessel. White thrombi generally form in those places where there is still some circulation of the blood going on.

The process of the formation of thrombi can be observed in the larger veins of the mesentery of the frog, if it be exposed so as to be placed under the microscope. At a given point you will observe that there is an accumulation of white blood-globules, not over the whole periphery of the vein, but at some particular part; these will be swept away by the circulation. Then there will be another collection of white blood-globules formed in the same way, and these, too, may be swept away by the passing blood; but finally, there will be a collection of white blood-globules which will remain, and will become larger and larger, until the entire vein is filled with a mass of white globules. Then, if you continue to observe, you will find the outlines of the white blood-globules becoming less and less distinct, and that you have a mass of coagulated fibrin entangling a certain number of white blood-globules. It seems probable that this is the way in which a certain number of ordinary thrombi are formed in the human subject.

When thrombi are formed they undergo certain changes. If they are of a red color, the first change which they undergo is to become decolorized, so that they resemble in appearance thrombi which were at first white. If the patient continues to live, these thrombi in some instances become organized, being replaced by permanent new connective tissue containing blood-vessels. When this takes place, the particular blood-vessel in which it occurs is permanently obstructed. In still other cases the thrombus, instead of becoming organized, becomes calcified; the fibrin becomes infiltrated with the salts of lime, and then we have the calibre of the vessel occupied by a hard white mass. In still other cases the thrombus degenerates and becomes softened; becomes more brittle. Part of it is converted into a thick reddish fluid, and the parts which continue solid become brittle, so that portions of them may be detached and pass into the general circulation. This softening is sometimes merely a simple process of degeneration; at other times the degeneration is accompanied with the production of bacteria, in which case the process becomes a somewhat more complicated one, and more dangerous to the life of the patient. When a thrombus softens, and there is also a production of bacteria, then the fragments that become detached will act as irritants in the different parts of the body into which they go, and will there set up an inflammation. In the other case they do harm only by their mechanical presence.

There is a marked increase in the severity of the constitutional symptoms accompanying the degeneration of a thrombus in either of the ways I have mentioned, and the walls of the vein are very apt to become inflamed and infiltrated with pus-cells. In extreme cases this may be sufficient to perforate or destroy a part of the vein.

Causation.—In order that blood should not coagulate, and should circulate, two things are necessary: *First*, the blood must be able to be in constant motion; there must be no mechanical obstruction to its passage through the arteries, veins, and capillaries. *Second*, the condition of the inner surface of the walls of the blood-vessels must be perfectly normal. The moment we have a departure from either of these two conditions it is possible for thrombosis to occur. In some instances we have thrombi formed in the veins apparently spontaneously. This is especially the case in the veins of the neck, legs, pelvis, duramater, and the capillaries of the toes, and in the abdominal aorta. In these cases, though we call the thrombosis spontaneous, yet the changes in the circulation of the blood exist, and these are often due to the general condition of the patient. A patient may be so enfeebled by disease, or his heart may be working so inefficiently, that the blood is not passed from it into the arteries, from the arteries into the capillaries, and from the capillaries into the veins, as rapidly as it ought to be.

If an artery or vein is compressed by a ligature, or by a tumor, or by the dislocation of a bone, then there exists a mechanical obstruction, and a thrombus may be formed as a result of it. If either of the cavities of the heart are dilated, or arteries or veins are dilated, we have a condition favorable to the formation of a thrombus. We may have an aneurism of an artery, or a dilatation of a vein, giving rise to a change both in the circulation of the blood and the inner surface of the arteries or veins. Should the tissues around a vein become inflamed, the process may extend to the wall of the vein producing a change in the circulation of the blood, and in this way give rise to the formation of a thrombus. Again, in the heart, arteries, and veins there are chronic inflammatory processes which affect the walls of these vessels, and as a result we may have thrombi formed. These are the ordinary circumstances under which thrombi have their origin.

Results of Thrombosis.—The results of the formation of thrombi vary with their particular position. When formed in the heart they may interfere with its action; detached portions may be carried into the circulation, and plug a certain number of arteries or capillaries. When thrombi are formed in the arteries, they commonly produce slow death of the particular part of the body which is supplied by that artery. The part becomes anæmic, gradually dies and degenerates. Thrombi may give rise to inflammation of the veins; they may produce œdema of the particular part of the body to which the vein belongs, or they may produce gangrene, or hypertrophy of this part, or there may be extravasations of blood into it. When thrombi are formed in the capillaries, they produce gradual death of the part.

OF EMBOLISM IN GENERAL.—Now we come to the process of embolism. An embolus is a solid body, most frequently a portion of a softened thrombus, which has passed into the circulation and floats in the blood, and is propelled by the action of the heart into the arteries and capillaries. If the embolus is of considerable size it will completely occlude the vessel into which it is forced by the action of the heart; if it is small, it may only partially occlude the vessel. In other cases, after it has become lodged, a thrombus will form about it, and in this way the occlusion of the vessel is usually made pretty complete.

Causation.—The ordinary causes of embolism are, in the first place, the presence of thrombi in the veins. If thrombi are situated anywhere in the veins, and they degenerate, soften, and break down, the parts becoming detached pass into the blood-current and are carried to the right auricle of the heart. The contraction of the right auricle drives them into the right ventricle, and from thence they are forced into the pulmonary artery, and may then pass into the capillaries of the lungs. If they are of a certain size they become fixed either in the arteries or capillaries. If sufficiently small they pass through the capillaries of the lungs, thence into the pulmonary veins and to the left auricle of the heart; by the contraction of the left auricle they are driven into the left ventricle, from thence they pass into the aorta, there to become fixed in some of the small capillaries belonging to the aortic system.

In other cases these emboli are formed from thrombi situated in the cavities of the heart. Should a thrombus be in one of the right cavities of the heart it may pass into a branch of the pulmonary artery and there become fixed. If a thrombus is situated in one of the left cavities of the heart, and parts of it become detached, they pass into the aortic system of vessels, and finally plug one of the branches of that system. In some cases we have disease of the valves of the heart, or of the endocardium of the heart, and, as a result, we have the formation of what are called *vegetations* of the valves, or of the endocardium. These vegetations are practically nothing but thrombi; nothing but coagulated blood; or, in part, they may be formed of new tissue, which may have the same structure as that of the valve or endocardium. When parts of these become detached, they act as emboli. Similar changes take place in the walls of the aorta, producing vegetations which may become detached and pass into the general circulation, acting as emboli. Sometimes we have tumors projecting into the cavities of the veins, and fragments of these may become detached and act as emboli. Then there are still other cases in which we find emboli in one or more arteries, and yet we are unable to determine from whence they came. They must always be derived from a thrombus situated somewhere, but we may be unable to find out the location of this on account of the large number of veins, so that there are a certain number of cases in which the origin of the emboli cannot be found.

Results of Embolism.—If an embolus becomes lodged in an artery of some little size, and if this artery is provided with numerous anastomotic branches, the patient feels a certain shock at the time the embolus becomes lodged: he may feel some pain in the part of the body where the artery is situated; there may be, for a short time, for a few minutes, or a few hours, a change in the temperature and color of the part, but after this the anastomosing arteries supply the part with enough blood to nourish it as well as before, so that no permanent symptoms are produced. The pain passes off, the diminution in the temperature of the part no longer obtains, the temporary anæmia fades away, and the patient ceases to be aware that anything unnatural is going on in his blood-vessels; but still the particular artery which was plugged never returns to its normal condition. A thrombus forms around the lodged embolus, and the whole is gradually replaced by new connective tissue. The patient does not suffer on account of this, for the circulation is carried on by the collateral arteries.

On the other hand, if the embolus becomes lodged in an artery which is not provided with very large or very abundant anastomosing branches, its lodgment is marked, as before, by a certain amount of shock and pain in that part of the body, by anæmia, and fall of temperature. But instead of the patient soon becoming comfortable, he continues to suffer; for the part which was supplied by this artery, although still receiving a little blood through the imperfectly anastomosing vessels, does not get enough blood to nourish it, and as a result of this it slowly dies: slowly undergoes necrotic changes. If it is an artery of one of the extremities which is plugged in this way, then that extremity will gradually undergo gangrene. We see this not infrequently in the leg after embolism of the popliteal artery; the leg below the embolus gradually dies, becomes gangrenous, and a line of demarcation forms. If it is a smaller artery which is occluded then a smaller part of the tissue will in the same way die, or undergo necrotic changes. We observe these changes most frequently in the brain, skin, and extremities; especially in the legs.

INFARCTIONS.—When an embolus becomes lodged in an artery not described as having anastomosing branches, the result of the embolism is *infarction*, and of this there are two kinds, *hemorrhagic* and *white* infarction.

Hemorrhagic Infarction.—The hemorrhagic infarctions are formed by the congestion of the part, and by extravasations of blood into the part. Perhaps the simplest example of this sort of infarction is the plugging of the renal artery. We know that the kidneys are supplied by an artery and vein. If now the artery is plugged the result of this is not a permanent anæmia of the kidney, or a gradual death, but it is the congestion of the kidney; it becomes gorged with venous blood. We are able to see this as often as we wish by experimentation upon the lower animals. If we ligate the renal artery of a dog, or a rabbit, we find that the kidney is the seat of venous congestion, and there is also extravasation of blood into the tissues. The same condition obtains in the human subject as a result of plugging terminal arteries in different parts of the body. For example, if we have an embolus carried into the lungs, and it becomes fixed in one of the smaller arteries, we find, as a result, that a circumscribed portion of the lung, usually of a peculiar wedge-shape, and corresponding to the area of tissue traversed by the artery, set of capillaries, and the vein beyond the plug, is congested; and that, at the same time, there is circumscribed extravasation and infiltration of blood. Such an infiltration of blood, as the result of the plugging of a terminal artery, is called a hemorrhagic infarction.

Virchow's Theory of Infarction.—The explanation which was first given to this condition was, that both the congestion of the part, and the extravasation of blood into it, were due to a regurgitation of the blood. It was supposed that congestion of the kidney, after plugging of the renal artery, was due to a regurgitation of the venous blood through the veins into the kidneys. This explanation was advanced by Virchow. It became evident that, although this theory would account for the venous congestion, it would not account for the extravasation of blood into the tissues. There was no reason why, with the existence of a plug in the artery, there should be greater pressure upon the blood here than in any other vein; it was not easy to understand why there should be any extravasation.

Cohnheim's Theory.—To account for this it was further supposed by Cohnheim that a change took place in the walls of the congested blood-vessels of the part. He found that if ligatures were applied for a short time to terminal arteries, so that the system of vessels was for a short time emptied of blood, and the ligature then removed so that the blood could return, the result was an extravasation of blood resembling an infarction. He inferred that the plugging of the renal artery produced a change in the walls of its branches and capillaries, and in consequence of this the blood passed through them and became infiltrated into the tissues.

More Recent Investigations.—The explanation has rested here for a considerable time; but now our minds are still further disturbed. No changes in the walls of the vessels can be demonstrated, and, in addition, it has been found that, if the artery and vein be ligated, the same result is obtained as when the artery alone is ligated; so we cannot account for all of the phenomena on the ground that there is a regurgitation of venous blood and a change in the walls of the blood-vessels.

By separating the capsule of the kidney from that organ, or by putting a ligature around the loose tissue surrounding the capsule, it was found that the picture of hemorrhagic infarction was no longer repeated; the same thing was found in the lungs. If, in addition to the ligation of the artery and vein, the bronchial and pleural arteries were ligated, the lung would become anæmic. These experiments seemed to show that it was necessary to take into account that the kidney had a capsule which was supplied with blood-vessels that passed into the substance of the kidney, and that this capsule was supplied with other blood-vessels which anastomosed with still other vessels of the connective tissue.

So, when the renal artery and vein are ligated, it is found that the kidney is not cut off from the general circulation of the body, but blood passes into it from the outside arteries, which communicate with the arteries of the rest of the body, and thus blood is extravasated into the tissues of the kidney.

There are enough arteries running into the cortex of the kidney to supply it with a certain amount of blood; but the renal artery being ligated, there is not sufficient impetus given to the blood to cause it to be carried off by the capillaries and veins. In addition, we have regurgitation of blood into the vein, which requires the full force of the arterial current to move it on. As this force is taken away by the plugging of the artery, we have congestion of the kidney. These, then, are three explanations of the manner in which hemorrhagic infarction takes place.

Subsequent Changes.—When a hemorrhagic infarction has once formed, it regularly undergoes certain changes, and these are of a degenerative character. If the patient continues to live, the first change which takes place is that the blood loses its coloring matter, at the same time the infarction becomes somewhat harder and drier, and the part, though still infiltrated with blood, begins to assume a yellow color. Simultaneously there is an inflammatory process taking place in the zone of tissue immediately surrounding the infarction, so that, after the infarction has lasted for a short time, it ceases to be congestive, and is complicated by the changes in the tissues immediately surrounding it.

In a certain number of cases, if the patient continues to live, the processes of decoloration and drying go on, and finally the infarction becomes very much smaller than it was at first, being a small, dry, cheesy mass, composed of nothing but degenerated tissue, while around it may be found a little new connective tissue which has resulted from the inflammatory process to which I have referred. So, if the patient recovers, the portion of lung, kidney, or spleen which was the site of the hemorrhagic infarction, is a cheesy mass surrounded by a little new connective tissue.

In most instances hemorrhagic infarctions break down without passing through the stages which have been described, and they come to look like abscesses—cavities filled with a fluid resembling pus; but it is simply serum holding in suspension a certain amount of granular matter and broken-down tissue. This is what becomes of hemorrhagic infarctions when once formed.

White Infarction.—White infarctions, as they are called, are much more simple. We find them in a certain number of cases of embolism of the terminal arteries, most frequently in the kidneys and spleen. They are formed in this way: a small terminal artery is completely obstructed, resulting in anæmia of the tissues supplied by that artery. This anæmia is permanent; it is not succeeded by congestion. There is no regurgitation of venous blood; there is no supply of blood from any other source. As a result of this the tissue dies, it degenerates, and is converted into granular matter.

If the patient lives, it becomes smaller and harder, and finally presents itself as a small, white mass, composed of granular matter. If the patient dies, we find after death a small portion of tissue in different stages of degeneration, according to the length of time elapsed since the lodgment of the embolus. These, then, are the results produced by the lodgment of emboli in arteries.

Emboli producing Local Irritation.—The emboli we have been speaking of are those which do harm by their mechanical presence in the particular vessels in which they become lodged.; but besides these we have emboli which act as *local irritants*. Such emboli are derived from thrombi which have broken down in the putrid way. You remember, in speaking of thrombosis, I told you that a certain number of thrombi softened, broke down, and became fragmentary; and that, in addition to this, the process might be attended with putrefaction and the formation of bacteria. When thrombi of this sort are lodged in the circulatory apparatus, they do harm, not only by mechanically obstructing the circulation, but by setting up a local suppurative process, so that a small abscess is formed in the tissue immediately surrounding the irritating embolus. These abscesses are found most frequently in the lungs, less frequently in the kidneys and spleen, and sometimes in the liver.

LECTURE IX.

THROMBOSIS AND EMBOLISM OF THE BRAIN.

Changes produced by the Plugging of the Cerebral Arteries.—Symptoms of Thrombosis and Embolism of the Cerebral Arteries.—Occurrence and Cause of these Cases.

CHANGES PRODUCED BY THROMBOSIS AND EMBOLISM IN THE ARTERIES OF THE BRAIN.—*Gentlemen:* At our last lecture we spoke of thrombosis and embolism; we also explained to you what was meant by infarction, and how it was supposed to occur.

To-day we come to the consideration of the changes which are produced in the brain by the processes of thrombosis and embolism. In the brain we have veins, arteries, and capillaries. The arteries may be occluded by emboli or thrombi, the capillaries by emboli, the veins and sinuses only by thrombi.

Thrombosis of the cerebral arteries is most common in the internal carotids, in the middle cerebral arteries, in the basilar artery, and in the vertebral arteries, in the order named. In the other cerebral arteries they are found less frequently. We may find only a single artery occluded by a thrombus, or we may find the process going on at the same time in several arteries.

The walls of the artery which is the seat of thrombosis are very frequently thickened, or their inner surface roughened; in other cases, however, we are not able to make out any change in the walls of the vessel. We regularly find changes in the parts of the brain supplied by the artery or arteries occluded. Such a part of the brain first degenerates, then softens. The softening may or may not be accompanied by extravasations, which are either of a red color, or white, or yellowish. If the patient lives for some time after obstruction of the artery has taken place, the softening goes on until the brain-substance has fairly changed into fluid, and you have the appearance of a cavity filled with diffluent matter looking somewhat like pus, but it is degenerated brain-tissue floating in serum.

This condition is found more frequently in males than in females. It is caused sometimes apparently by changes in the walls of the blood-vessels, by pressure upon the walls of the blood-vessels, and it seems in some cases to depend upon the general condition of the patient, upon a lowered circulation, the blood not passing through the arteries with sufficient rapidity.

The symptoms of thrombosis in cerebral arteries are essentially the same as the symptoms of embolism in cerebral arteries, so we will not describe them separately, but together.

Embolism of cerebral arteries acts in two ways. We have small emboli in cases of pyæmia, and these produce little abscesses in the brain-tissues; but, inasmuch as these lesions enter so largely into surgical pathology, we do not have to consider them now.

The lesions which we have to consider are produced by emboli obstructing one or more of the cerebral arteries. Emboli of this kind have been found in nearly all of the cerebral arteries; but there is one pair which is especially apt to become the seat of such emboli, namely, the middle cerebral arteries, and of these the left is much more frequently obstructed than the right.

The result of the obstruction of a cerebral artery by an embolus is the same as the result would be from plugging a similar artery by a thrombus—that is, a circumscribed portion of brain-tissue becomes deprived of its nutrition, in consequence of which it degenerates, becomes softened, and finally breaks down into a diffluent mass. This is the ordinary result of the lodgment of an embolus in one of the cerebral arteries. There is one exception to this, however, but fortunately it is rare. There are cases in which the patient suffers from lesions of the aortic valves, in which there are extensive vegetations, which may be, for reasons unknown to us, of a peculiarly soft, crumbling nature. Sometimes a large mass of these vegetations may be swept away and carried to the brain, and if so, a large number of vessels may be obstructed at once. When this takes place the patient dies almost instantly; there is no time for the softening of the brain-tissue. This is one of the causes of very sudden death. A patient may be sitting in a chair, or standing, and will fall down suddenly and die almost instantaneously.

Symptoms produced by Plugging of Cerebral Arteries.—Now, as to the symptoms produced by plugging of cerebral arteries, whether by emboli or thrombi. We must consider, in the first place, what happens at the time of lodgment, or within a few minutes, or few hours after the lodgment of an embolus. In a certain number of cases the lodgment of an embolus is marked by the sudden loss of consciousness on the part of the patient, and this is accompanied by hemiplegia or loss of voluntary motion upon one side of the body, and is attended with coma, which lasts a longer or shorter time. This takes place when an artery of some size is blocked up; the patient at once becomes unconscious, hemiplegic, and comatose.

In other cases the lodgment is not attended with such marked symptoms; there is no loss of consciousness, but there is sudden hemiplegia. In still other cases the symptoms are slightly less marked. The patients not only do not lose consciousness, but do not lose the power of motion in all of the muscles upon one side of the body; they simply lose the power of motion in the muscles of the face, or of one arm, or one leg, or they become aphasic—they lose the power of speech. The cases in which the symptoms are not so severe are those in which one of the smaller branches of a cerebral artery is plugged. The loss of speech may last only for a moment, or for a longer time. The loss of voluntary motion may not be complete. If the patient comes to have hemiplegia, it may not be absolute; he may be able to move the arm or leg a little. If the muscles of articulation are involved, the patient may still speak, but with difficulty.

During the first twenty-four hours after the lodgment of an embolus we may have convulsive movements of some of the voluntary muscles. These convulsive movements take place in those muscles which are afterward to become paralyzed; in other words, an embolus acts as an irritant at first, giving rise to convulsive movements of one side of the body, or of one leg, or of an arm, together with convulsive movements of the muscles of the face; these movements, lasting for a moderate length of time, are succeeded by more or less complete paralysis of the affected muscles. Most frequently the patients have general epileptiform convulsions within twenty-four hours after the lodgment of a thrombus. The patient may continue comatose, may not regain consciousness at all, and may die at the end of a few days, or within the first twenty-four hours. Other cases do not die in this way, but go on to have permanent hemiplegia either with or without permanent aphasia; the loss of voluntary motion upon one side of the body lasts as long as the patient lives. The hemiplegia is usually upon the right side of the body, corresponding with the fact that it is most frequently the left cerebral artery that is plugged.

In those cases in which the hemiplegia and the aphasia is to be permanent, there is very frequently during the second, third, or fourth day after the plugging of the artery, a temporary amelioration of the symptoms of paralysis and aphasia. The patient may be able to move his arm or leg a little better, or speak with greater facility, but this improvement is only temporary, lasting but a few days, after which the paralysis and aphasia return and continue during the rest of the patient's life. This temporary improvement seems to correspond to changes which are going on in the brain. At the moment when an artery is plugged the part of the brain supplied with blood by it is deprived of nutrition. After the patient has lived for a few days the brain seems to accustom itself to the loss of blood, and there is a little return of power to the paralyzed muscles; but after the lapse of a few more days the degenerative changes take place, and the loss of power becomes permanent. This is what occurs in a certain number of cases.

In some cases of permanent hemiplegia and aphasia there is no disturbance of the mental functions except the loss of language, which may continue during the remainder of the patient's life. In other cases the intelligence is no longer as good as it was before the lesion was formed. The patients are not able to attend to business, and their memory is not good; they may forget their friends and relatives. In other patients again, after the hemiplegia is fairly established, we have chronic meningitis developed, which is attended with the symptoms belonging to it. Such patients may have an irregular febrile movement; they may suffer from a pretty marked localized pain in the head; they may become delirious, have convulsions, and finally die from purely chronic meningitis developed secondarily.

In those cases where the hemiplegia is permanent, no matter what the mental condition may be, if the patients live the muscles are very apt to become contracted. If, for example, the right arm and leg are paralyzed, after remaining so for a certain length of time you will find the arm is gradually being flexed, that the fingers are flexed upon the hand, and that the leg is being flexed in the same way. This contracture of the muscles goes on, becoming more and more marked as long as the patient lives.

Then there are still other cases in which the paralysis and aphasia gradually get better. This is especially apt to be the case when, at the very first, the paralysis and aphasia are not complete, but partial.

The prognosis is always better if at the commencement of the disease the paralysis and aphasia are only partial. When this is the case, after some weeks there will be a gradual improvement on the part of the patient; the paralyzed muscles will slowly regain more and more power, until finally the patient is able to use them as well as before the disease was developed. In the same way the aphasia gradually gets better, and the patient is able to say more and more words, or parts of words, until he speaks as well as before, or there may remain a few words which he remembers and speaks with difficulty. If the difficulty in speech is dependent upon a change in the muscles of articulation, this will improve, and the patient will speak with more and more ease as time goes on.

Then there is still another set of cases in which, after the patients have recovered from partial or complete hemiplegia and aphasia, they will have other attacks. This, unfortunately, quite frequently happens; it is one of the bad features of embolism of the cerebral arteries. There is a probability that the patient will have a number of such attacks, and the later ones are apt to be worse than the first, so that the results of these are apt to be worse and worse.

LECTURE X.

THROMBOSIS AND EMBOLISM OF THE BRAIN — CONTINUED. — LOCALIZATION OF BRAIN LESIONS.

Differential Diagnosis of Cerebral Embolism from Cerebral Apoplexy.—Localizations of Brain Lesions.—Lesions of the Corpora Striata.—Lesions of the Cortex of the Brain.

GENTLEMEN: We were last considering the effects of plugging of the cerebral arteries by emboli or thrombi. We will now go on to consider the diagnosis of this lesion of the arteries of the brain.

Diagnosis.—The diagnosis of cerebral embolism from that of cerebral apoplexy in a certain number of cases can hardly be made. It will be difficult in some cases to say whether the patient is suffering from plugging of a cerebral artery or from extravasation of blood in the brain, for the symptoms may be identical in the two cases. There are, however, some considerations which help us to make a diagnosis. In the first place, there is the age of the patient. Generally speaking, cerebral apoplexy occurs in persons comparatively young, and cerebral embolism in those who are quite old. This rule, though of use in making a diagnosis, cannot be relied upon to any very great extent, on account of the exceptions.

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If we find that the hemiplegia is upon the right side, and at the same time the patient is aphasic, then the probabilities are usually in favor of cerebral embolism. If the paralysis is upon the left side, and there is no aphasia, then the probabilities are in favor of cerebral apoplexy. If the patient only loses the power of the muscles of one arm, or the power of the muscles of the face, or only loses the control of the muscles of articulation, or if he becomes aphasic without the loss of voluntary motion, then this is in favor of embolism rather than apoplexy. In the cases in which hemiplegia occurs, if there is loss of consciousness it is likely to be apoplexy; if there is no loss of consciousness, then it is likely to be cerebral embolism.

If the symptoms remain for a short time; if the paralysis gets better after a day or so, and then becomes worse, this also points to cerebral embolism rather than to apoplexy.

The condition of the heart does not help us as much as one might think. Examining the heart, and finding no murmur, might lead one to think this pointed to apoplexy, but this is not the case. We find a great number of cases of cerebral embolism in which the heart is not changed, and in which there is no murmur. On the other hand, we find a good many cases of cerebral apoplexy with hypertrophy of the left ventricle, and this may or may not be accompanied with valvular lesions capable of producing a murmur at the same time.

Prognosis.—The prognosis of cerebral embolism varies in the different cases with the size of the artery which is plugged, and with the amount of brain-tissue which is consequently destroyed. Generally speaking, the cases in which the paralysis and aphasia are incomplete and the cases in which only certain groups of muscles are involved, are more favorable than those in which the hemiplegia and aphasia are complete. You will find, however, that it is always wise to wait for two or three weeks before giving any decided opinion as to the result of the case, for there is always a liability that it will be complicated by chronic meningitis. There are cases in which the symptoms are not as marked at first as afterward, and in which the immediate results of plugging of the arteries are not as bad as those produced by the degenerative and destructive processes. So that cases which seem to promise well at first may get worse instead of better; and some cases that promise badly at first, may, after a few weeks, begin to improve.

It is especially important to remember these facts, as the prognosis is really the most important feature of the case, so far as our relations to it are concerned, for the treatment amounts to nothing. The thing which the patient's friends, and which the patient himself is anxious to learn, is whether he is likely to remain permanently paralyzed, or whether he is likely to improve, and this you can never speak of with any certainty until after the lapse of some time.

LOCALIZATION OF BRAIN-LESIONS.—When there are lesions in the brain, no matter whether they be due to plugging of an artery or to the presence of a clot of blood, there is always an advantage in determining the precise portion of the brain in which the lesion is situated. This we can do to a certain extent. There have been a sufficient number of cases of cerebral apoplexy and embolism observed both during life and after death, for us to be able to speak with some certainty in regard to symptoms caused by such lesions in certain parts of the brain. I will give you a number of conclusions derived from the observation of cases before and after death, and I think you will find it to your advantage if you make a note of them now for reference in the future.

In the first place we have to remember that all cerebral lesions act in one of two ways: they act as irritants, or they act as destructive processes. If they act as irritants, the result of this is to cause convulsive movements in certain groups of muscles. If they act as destructive processes, the result is the paralysis of certain groups of muscles, and some lesions may act in both ways, first as irritants and afterward as paralyzers. This is true of softening of the brain produced by emboli or by clots. Either of these will at first cause convulsive movements of certain groups of muscles, which afterward may become paralyzed.

If there is much effusion of blood upon the surface of one or both hemispheres, as a rule it produces general convulsions.

If there is much extravasation of blood at the base of the brain, it usually causes the sudden death of the patient, sometimes almost instantaneously, sometimes at the end of a few hours.

Lesions of the Corpora Striata.—We come now to the different parts of the brain: in the first place of the *corpora striata*. The corpora striata are composed of two parts, the caudate nucleus, and lenticular nucleus, the relations of which you will be told. Now we find, that lesions which involve a considerable part of the corpus striatum, produce, as a rule, upon the opposite side of the body paralysis of motion and loss of sensation; sometimes paralysis of the vasomotor nerves.

If the lesion involves the lenticular nucleus of the corpus striatum alone, or if it involves the caudate nucleus of the corpus striatum alone, then the hemiplegia, as a rule, gradually disappears; but if the internal capsule is involved also, then, as a rule, the paralysis is permanent; it does not disappear. In these permanent cases of paralysis there is usually developed later contracture of the paralyzed muscles. Such hemiplegias involve regularly the nerves of the arm, leg, and the branches of the facial nerve upon the opposite side of the body from that upon which the lesion exists. The hypoglossal nerve is not usually involved, but if so, it is commonly only temporarily. In rare cases, only the muscles supplied by the facial nerve, or the muscles of the extremities are paralyzed.

Lesions of the lenticular nucleus occurring alone, cannot be distinguished by their symptoms from lesions of the caudate nucleus occurring alone. If the lesion involves only the anterior portion of the corpus striatum, there is only motor paralysis; there is no loss of sensation.

In a certain number of cases with lesions of the corpus striatum, there is complete anæsthesia of the skin, and in addition to this there is loss of sight, hearing, taste, and smell, on the same side of the body on which the hemiplegia exists. When there is such a loss of sensation in the skin, it usually points to changes in the internal capsule, in addition to the changes in the corpus striatum.

In some cases of lesion of the corpus striatum there is paralysis of the vasomotor nerves, and as a result of this the paralyzed side of the body is of a higher temperature than the opposite side, and there is persistent venous congestion. When this is the case, there are usually changes in the posterior part of the internal capsule, in addition to changes in the corpus striatum. These make up all the facts which we know with any degree of certainty of the lesions of the corpus striatum.

Lesions of the Cortex of the Brain.—When we speak of lesions of the cortex of the brain, we refer, as a rule, to the two central convolutions of the brain, as they are called, and to the part of these convolutions which is called the lobulus paracentralis, to the island of Reil and to the third frontal convolution. These are the parts of the cortex which we have to consider.

Diseases of the gray matter of the cortex of the brain, or rather lesions of the white matter immediately beneath it, in a certain number of cases produce symptoms, in other cases they are not attended with any symptoms at all. This is a matter of some consequence, that it is possible to have lesions of the cortex of the brain, which will actually produce no symptoms.

Disturbances of the intellectual faculties generally point to a diffuse lesion of the cortex, to such a lesion as we get in chronic meningitis, and to such a lesion as we get in the progressive paralysis of the insane. Disturbances of the faculty of speech, and disturbances of the muscles which control the articulation of words, usually indicate a lesion of the cortex, and the part of the cortex which is usually changed is the third convolution and the island of Reil upon the left side of the brain, where we find the lesion in the great majority of cases when there is aphasia.

When there is loss of hearing, there is in a certain number of cases a lesion of the left temporal lobe, especially of the first temporal convolution upon the left side.

When there is loss of vision in the half of each eye, there is, in a certain number of cases, a cortical lesion; but this can also occur with a lesion of the corpus striatum. If this symptom occurs without other cerebral symptoms, in a certain number of cases it indicates a lesion confined to the occipital lobe. When there is no lesion in the eye to account for it, there is always a probability that there is a lesion in the occipital lobe of the brain.

When there are disturbances of the sensibility of the skin, it is more frequently due to lesions of the corpus striatum than to lesions of the cortex.

If the patient suddenly becomes aphasic without loss of voluntary motion, or without loss of sensation, this points, as a rule, to a circumscribed lesion in the cortex, to a circumscribed lesion in the island of Reil, or the third convolution. This rule is one to which there are only a few exceptions. If a patient suddenly has paralysis of the upper eyelid upon one side without any other cerebral symptoms, this points, as a rule, to a circumscribed lesion in the cortex.

Whenever there is an isolated paralysis of an arm, or of muscles supplied by the facial nerve, or of parts supplied by the hypoglossal nerve, then there is usually a cortical lesion. The same is true of paralysis of any isolated group of muscles: it is always probable that the lesion, under such circumstances, is a cortical one, and not central.

If the patient suffers from convulsive movements, and if these are confined to groups of muscles which are afterward to become paralyzed, this is almost a certain sign of a cortical lesion.

This comprises all we know with any very great certainty of the cortical lesions of the brain as distinguished from central lesions.

LECTURE XI.

THROMBOSIS AND EMBOLISM OF THE BRAIN — Concluded. — ABSCESS OF THE BRAIN.

Thrombosis and Embolism of the Venous Sinuses of the Duramater.—Of Thrombi which Remain Hard.—Of Thrombi which Become Soft.—Abscess of the Brain; Location, Pathology, Duration, Symptoms, and Course of the Disease.

Thrombosis of the Venous Sinuses of the Duramater.—*Gentlemen:* We were considering at our last lecture the results of thrombosis and embolism of the arteries of the brain. We come to-day to the consideration of the results produced by thrombosis of the venous sinuses of the brain, or rather the venous sinuses of the duramater. In this situation occur thrombi of two kinds.

Thrombi which Remain Hard.—In some cases a sinus is filled with a thrombus which is perfectly firm, showing no disposition to soften or break down. Such a thrombus may fill a venous sinus for a considerable portion of its length, and the veins leading from the brain to this sinus will be found occluded with similar thrombi, or else filled with blood. If we examine the brain when fresh we find that the part of it from which these veins come is softer than the rest; and we also find a certain amount of extravasated blood, so that the softened portion of the brain is redder than it should be, on account of the admixture of blood.

Symptoms.—Thrombi of this kind give rise to cerebral symptoms, which vary with the extent of the softened brain-tissue, and the particular part which is thus affected. The lesion is not a very common one; it is on the contrary a very rare one. There are not enough cases upon record for us to know all the symptoms that can possibly arise from this condition. In the cases observed, the symptoms have been somewhat like those of apoplexy, that is, like those cases in which there is an extravasation of blood into the brain, or an embolism of a cerebral artery. The cases are more fatal, however, than those of embolism of a cerebral artery.

The attack is abrupt in most cases, although the thrombus is probably formed slowly; but the patients do not give us any symptoms attending its formation. The first symptom usually noticed is that the patient becomes suddenly unconscious, and this condition is accompanied with hemiplegia. The patient remains unconscious, becomes comatose, and finally dies comatose at the end of a few days. This has been the course of the disease as it has been observed. We have to suppose, that if smaller portions of the brain were involved and out of the motor track of the brain, we would have less marked symptoms; but so far as our experience goes, the symptoms above given are all that have been noted. They are identical with those of extravasation of blood in the brain, and the plugging of a large cerebral artery by an embolus.

Thrombi which Become Soft.—In other cases we find thrombi formed in the venous sinuses which do not remain firm and hard; they break down, and the softened parts, becoming detached, pass into the general circulation. As a result of this we sometimes have hemorrhagic infarction and abscesses in different parts of the body. The breaking down of thrombi seems to act as a local irritant to inflammation of the piamater. This process is ordinarily secondary to some other cause. It occurs frequently with suppurative inflammation of the internal ear, and we find it with different inflammations of the duramater, with fractures and inflammations of the cranial bones, with thrombosis of the veins of the neck, and with erysipelas of the face and scalp. Less commonly we find it as an idiopathic condition, there being no primary cause to account for its occurrence.

Symptoms.—Thrombi which soften give rise to symptoms of two kinds: those belonging to the meningitis, which the thrombosis sets up, and those due to the production of infarction and abscess in other parts of the body.

The symptoms due to the meningitis are the same as those of any case of acute meningitis, and hence it is not necessary for me to recapitulate them.

The symptoms produced by the lodgment of parts of the softened thrombus in different regions of the body are such as we meet with in pyæmia: first rigors, then an irregular febrile movement, rapid emaciation, and loss of strength, the patient coming to have a peculiar haggard appearance, and finally dying in a condition of extreme exhaustion. Thus softened thrombi give us a set of cases in which we get a combination of the symptoms of acute meningitis, and those of pyæmia.

ABSCESS OF THE BRAIN.

Location and Pathology.—Now we come to the consideration of a lesion of the brain-substance proper—to the consideration of abscess of the brain. Abscesses may occur in any part of the brain. They are found very frequently in the parietal and temporal lobes of the cerebrum. These abscesses are for the most part single, but it is possible for them to be multiple. Their character may be the same as that of an ordinary abscess, and have the same appearance as those found in connective tissue in any part of the body. A cavity is formed in the brain-substance and this contains pus. Its walls are ragged, being composed of shreddy, disintegrated brain-tissue; they are also infiltrated with pus. A microscopical examination of the pus found in very old abscesses, if it be particularly thin, will show it to contain hardly any healthy pus-globules. We find fatty pus-globules, granular matter, and cholesterine crystals. In very old abscesses the walls may take on the form of a fibrous capsule, being comparatively smooth inside so that it will look like a cyst of the brain instead of an abscess. The walls become thickened, and the pus, becoming thin, will resemble ordinary pus very little: it may change into serum.

In other cases the abscess does not look like an ordinary one, but has the appearance of a part of the brain-tissue being filled with pus. The magnitude of these differ a great deal. They are apt to be of considerable size, as large as a pigeon's egg in some instances, and not unfrequently very large indeed, replacing nearly the whole of the cerebral lobes. Occasionally the walls rupture, and this is especially the case if the abscesses are situated near the surface of the brain, or near the ventricles. When this happens we find the pus extravasated into the piamater or the ventricles of the brain. Abscesses situated very close to the surface of the brain frequently set up meningitis, so that the disease will be complicated by this affection. Sometimes they cause the production of thrombi in the venous sinuses of the duramater.

Etiology.—Abscess of the brain is frequently due to a definite cause. The most frequent causes are blows upon the head and inflammation of the middle ear. Blows upon the head, which are inflicted with considerable force, and yet which do not fracture the skull, are the particular injuries which are most likely to produce abscesses of this kind. Patients who have been beaten about the head, or who have fallen and struck their heads without giving rise to fracture, are those in whom, after a time, abscesses of the brain are developed. For a time, at least, no symptoms are produced; but after several weeks or months have elapsed, the symptoms of abscess of the brain begin to be manifest. It usually takes considerable cross-questioning to find out the cause of such abscesses, as sufficient time may have elapsed, since the date of the injury, for the patient or his friends to have forgotten it.

Some of you who were here last winter will perhaps remember a boy who came to the clinic with the history, that his teacher at school had beaten him with a long pointer, striking several blows with it upon the side of his head. After the lapse of some weeks the symptoms became well developed, and the case presented as a characteristic example of abscess of the brain.

Suppurative inflammation of the middle ear is also a tolerably common cause of abscess of the brain. Diseases of the cranial bones may give rise to the formation of such abscesses. Besides cases due to the above causes, we meet with some in which no reason can be found to account for their existence.

Duration.—The duration of the disease, so far as marked by symptoms, varies from a few days to a considerable number of years. Its average duration is only about two months, but the continuance of its existence always depends very much upon the situation and size of the abscess. There are parts of the brain, away from the motor tract and membranes, in which abscesses may exist for years and yet give rise to very few symptoms. Should abscesses thus situated increase in size, so as to invade the motor tract, or approach the membranes of the brain, the symptoms become more marked. Whereas abscesses situated near the membranes of the brain, or the motor tract, will at once give rise to marked symptoms, which continue until the patient dies.

Symptoms, Course, and Prognosis of Abscess of the Brain.—Of the symptoms which belong to this disease, headache is a very constant one. It is of a dull and continued character, lasting for a long time: weeks or months. In the more acute cases it is so severe that patients are not able to bear it. They ask for morphine, or an anodyne of some kind to relieve them. Patients are usually drowsy, and from this condition they may pass into one of complete stupor. Drowsiness may alternate with delirium, especially if the case be complicated with meningitis; but delirium is present in some cases, and absent in others. The disease is apt to begin with a rigor if it runs an acute course; if it runs a chronic course, rigors may be altogether absent. If there are rigors, there is usually a febrile movement following, and patients will have a temperature of 103°, 104°, or 105°. This high temperature may continue during the whole course of the disease. In the more chronic cases we may have hemiplegia, if the abscess involves the motor tract of the brain. If, however, the abscess is of very considerable size, then we may have lôss of power on both sides of the body, and instead of simple hemiplegia there will be loss of power in the muscles of both legs. In some cases there are general convulsions. If the abscesses are situated so as to press upon, or destroy any of the cranial nerves, the functions of these will be interfered with. Patients in this way may become deaf, lose their eyesight, or the sense of taste, or smell, or there may be loss of voice according to the particular nerves that are involved by the abscess. If the abscess is situated in the left lobe in the island of Reil, or in the third frontal convolution, these patients will become aphasic, or lose the power of speech.

The termination of cases of acute abscess of the brain is fatal, the patients dying from the presence of the abscess. In chronic cases the patients not infrequently die of some intercurrent disease, the abscess not being in itself a fatal lesion. Some of these abscesses perforate outward, and pus is extravasated beneath and into the piamater. In consequence of this, meningitis is set up, and patients have the symptoms of acute meningitis in addition to the symptoms of abscess of the brain. If the abscess penetrates into the ventricles of the brain the patients, at the time of the perforation, usually have an attack of convulsions, which is succeeded by delirium, which in turn passes rapidly into coma, and the patients die comatose.

LECTURE XII.

LESIONS OF THE VENTRICLES OF THE BRAIN. — HYDROCEPHALUS. — TUMORS OF THE BRAIN.

Acute Inflammation of the Ependyma; Symptoms.—Chronic Inflammation of the Ependyma; Symptoms.—Chronic Hydrocephalus, Congenital Variety; Pathology, Symptoms, and Course of the Disease.—Second Set of Cases, Mild or Severe.—Symptoms and Course of the Disease in Mild Cases.—Symptoms and Course of the Disease in Severe Cases.—Treatment.—Hydrocephalus, not Primary; Symptoms and Course of the Disease.—Other Cases of Hydrocephalus; Pathology, Etiology, Occurrence, Symptoms.—Tumors of the Brain; Character, Symptoms, Duration.—Recapitulation.

INFLAMMATIONS OF THE EPENDYMA.—*Gentlemen:* We come now to speak of some of the lesions which affect the ventricles of the brain. Those which are especially the seat of these lesions are the lateral or the chief ventricles. The fourth ventricle is also sometimes involved by disease. The ventricles, as you know, are normally lined with a thin membrane, called the *ependyma*, which is formed of connective tissue, and whose free surface is covered with flat cells. It is rarely the seat of acute inflammation, although we do meet with such cases. It is more liable to chronic inflammation.

Acute Inflammation of the Ependyma.—When acutely inflamed, we find the ependyma congested; it contains more blood than it should. The ependyma and the layer next to it are infiltrated with pus-cells, and along with these there is a certain amount of extravasated blood. The surface of the ependyma is coated with a layer of fibrin and pus, and in the cavity of the ventricles there is a certain amount of purulent serum. The rest of the brain may be entirely normal; this is true in the uncomplicated cases. In those that are complicated, however, there may be, at the same time, acute meningitis and acute inflammation of the ependyma. The dura around the ventricles may be softened to a certain extent. There are uncomplicated cases in which the ependyma is alone involved; but these are quite rare.

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Symptoms.—Patients who suffer from both acute meningitis and acute inflammation of the ependyma manifest the ordinary symptoms of acute meningitis; but when the piamater is not involved the symptoms are somewhat different. As an example of the symptoms and course of the disease, I will merely give you the history of a single case in which only the ependyma was affected.

The patient was an old man, about seventy years of age, whose health had been good enough until about two weeks before his death. His friends first noticed that his mental faculties were not as good as they had been; that his memory was failing; that he did not notice things about him; that his mind was becoming obscured. He continued in this condition until nine days before his death, when he became very stupid indeed, and very drowsy, so that if left to himself he lay in bed apparently half asleep, taking no notice of anything about him. Seven days before his death there was noticed paralysis of the right arm. This was only temporary, and disappeared in a day or so. The man remained in this same stupid and drowsy condition, with no other symptoms, until about two days before his death, when he became fairly comatose, and remained so until the night before he died, when he had a single convulsion, and finally died comatose. These were all the symptoms: a progressive stupidity, drowsiness, temporary paralysis of one arm, comatose condition followed by a single convulsion, and death in a comatose condition. These are the symptoms of acute inflammation of the ependyma of the brain.

Chronic Inflammation of the Ependyma.—Besides these rare cases of acute inflammation of the ependyma, we meet very much more frequently with those which are apparently chronic; but we have no very good evidence of the existence of chronic inflammation of the ependyma. We find in these supposed chronic cases that the ventricles of the brain contain more serum than normal, and that the ependyma is changed, it always being thicker than it should be. Sometimes its inner surface is changed by new growths of connective-tissue, and the thickenings occur in plates. These are all the changes we can find, and they are similar to those we are accustomed to think of as due to chronic inflammation. We must confess, however, that it is different from the chronic inflammation we usually meet with; it is more like dropsy; nevertheless, the course of the disease is such that we are disposed after all to look upon the lesion as one due to chronic inflammation.

This lesion of the brain presents itself to us in three sets of cases, each of which gives a very different clinical history, although the lesion is the same. One of these is developed in very young children; another occurs in children and adults as a sequence of certain chronic inflammations of the piamater; and in the other, it is developed in adults as an idiopathic lesion.

CHRONIC HYDROCEPHALUS; CONGENITAL VARIETY.—In the first place, of the cases that occur in very young children. These are commonly known under the name of *chronic hydrocephalus.* They are very frequently called ***chronic internal hydrocephalus***, to distinguish them from ***acute hydrocephalus*** (tubercular meningitis) or from ***external hydrocephalus***.

Pathology.—In very young children, if the disease proves fatal, there is a lesion of considerable extent. The lateral ventricles are enormously dilated, and this dilatation involves, in addition, not only the lumen of the canal extending from the third to the fourth ventricles, but very often the central canal of the spinal cord. The distention is so great that the brain-tissue is flattened against the duramater, and in consequence of this the convolutions and the sulci are partly obliterated. The ependyma is usually thickened, and its free surface is sometimes roughened. Occasionally there is softening of the brain-tissue adjacent to the ventricles. The distention of the brain by fluid in the ventricles is, in most cases, sufficiently great to expand the duramater and the cranial bones. In this way the whole cranium is enlarged, so that while the face remains the same as that of a child of a corresponding age, the head and cranial cavity become very much larger. The lesion occurring, as it does, at a time when the cranial bones have not united, we have the fontanels and sutures very much enlarged. Apparently, in these cases, the disease begins during intra-uterine life. The time at which the lesion becomes sufficiently pronounced to attract attention varies. This stage of its development may not be reached until several months after the birth of the child.

Symptoms and Course of the Disease in Severe and Mild Cases.—The symptoms vary with the rapidity of the development of the lesion. If it is developed very rapidly indeed, it is apt to have reached such an extent by the end of pregnancy that the child cannot be born alive on account of the head being too large to escape from the pelvis of the mother in the ordinary way. When such a condition exists, some operative procedure is necessary; this usually consists in the perforation of the cranium, its crushing, and the extraction of the child. Occasionally, in women with very large pelves, such a child may be born alive, but it will live only a few hours. This is what happens when the lesion develops rapidly during fetal life. If the child is born alive, cerebral symptoms show themselves shortly after birth. It is noticed immediately that the head is too large; the child does not appear as lively as it should. It does not eat well. After a few days convulsions come on, and these may continue for a limited number of days, when the patient will die. Or, after developing convulsions, it may pass into a condition of stupor, remain in this state for a few days, or a few weeks, and then die. During all this time the child does not eat as well as it ought, and it finally dies, partly from inanition and partly from the exhaustion due to the disease.

Then there are cases in which the lesion is very slightly developed during intra-uterine life. The child at the time of its birth appears as well and healthy as any child The head is not too large, and all of the functions are properly performed. After the lapse of weeks, or months, its head begins to enlarge; the size of the sutures and fontanels gradually increases, and there will be changes in the general condition of the child. These cases are mild or severe.

Mild Cases.—In the mild cases the intelligence will continue perfectly good; the mental faculties will be as well developed as in a child of the same age. It will learn to talk, will take as much notice of surrounding objects, and will be apparently as healthy as any child. From time to time, however, it will suffer from disturbances of digestion. These are accompanied by impairment of nutrition, and sometimes with a moderate febrile movement, and with ill-defined cerebral symptoms. Now and then the child becomes constipated, it will not take food, and may occasionally vomit. The parents or friends will notice that its head is hotter than normal; and with the thermometer you may determine the existence of a slight febrile movement. The child now rapidly loses flesh, so that in a few days there is a noticeable reduction in the size of its limbs; the muscles become soft and flabby; the temper of the child is changed: it is irritable and restless, and has fits of crying without any special cause. At other times it is unnaturally drowsy. The child takes less food for a few days or a few weeks, after which these symptoms may gradually subside, and the child will begin to eat. It will have a natural temperature, sleep well, and continue to get better gradually, gaining in flesh and strength, and finally return, with the exception of the abnormal size of its head, to a natural condition. Such children do not usually die from the lesion of the brain, but from some intercurrent disease. They not infrequently live to the age of twenty, thirty, or forty years.

Severe Cases.—In the severe cases, however, the symptoms are more pronounced. There are no such periods during which the child is apparently entirely well. The large size of the head is more noticeable, the distention of the ventricles being greater. The intellect is also impaired. These children are often not as bright as other children, and in the worst cases they are idiots. From the time that the disease is fairly developed their nutrition is disturbed. They are thin and pale, and are troubled with disturbances of the stomach and bowels. Quite frequently they will have internal strabismus. The eyes may be continually moving to and fro. The pupils are apt to be constantly dilated. Marked protrusion of the eyes may obtain, owing to the pressure of the dilated brain upon the orbits; this may reach such an extent that the eyelids will be partially closed behind the eyes, and assistance will be required to again get them in front of the eyes. Some patients lose their eyesight altogether; others become deaf, and the deafness may be quite complete. If the loss of hearing occurs at a very early age, children do not learn to talk. Not infrequently they have convulsions at intervals of days, weeks, or months; sometimes they become fairly paraplegic, losing the power of motion in both legs; they may continue suffering from these symptoms to a greater or less extent for months or years. They do not suffer to an equal degree at all times. Even in the bad cases there are periods when the children seem better, but they never appear to be entirely well. A number of these children finally die in an attack of convulsions; some die apparently from a lack of nutrition; others from intercurrent diseases. In the bad cases it is exceptional for patients to live until adult life; they more frequently die from convulsions, starvation, or some intercurrent disease while still children.

Treatment.—In regard to the treatment, there is very little to be said. There was a time when considerable attention was paid to the treatment of these cases, especially the mechanical treatment. Compression of the head and evacuation of the fluid was resorted to; but none of the plans of treatment adopted were attended with any apparent success, and are no longer used. The best we can do is to provide for the nutrition of the patient; we can do nothing for the head proper.

The nutrition of the child can be improved to such an extent that in mild cases the condition of the patient will be materially benefited. In bad cases such treatment amounts to but very little.

HYDROCEPHALUS, NOT PRIMARY.—We come now to that form of dilatation of the ventricles which we meet with in older children and in adults as a secondary condition. It occurs as a consequence of cerebro-spinal meningitis, and of acute meningitis due to any cause; as an accompaniment of chronic meningitis, and as the result of severe injuries to the head. In these cases the inflammatory lesion is more marked than in the congenital cases of which we have just been speaking.

Symptoms and Course.—If the disease follows an attack of cerebro-spinal meningitis, or an attack of acute meningitis due to any cause, the case runs through the regular course of the primary disease; but, instead of the patient getting well, he continues to have, though to a less extent, an irregular febrile movement. The patient's mental condition does not return to the normal; he remains delirious, stupid, and drowsy. There is no improvement in his nutrition; but, on the contrary, he continues to emaciate. Thus the case may go on for weeks, occasionally for months, until the patient dies from exhaustion.

When you make the post-mortem examination, you find that the piamater does not show the acute lesion of the primary disease, but you find the ventricles of the brain dilated and the ependyma more or less thickened. The ventricles are filled with a fluid that is at times quite clear, or it may be turbid from admixture of pus-globules. When the disease occurs as an accompaniment of chronic meningitis, the symptoms are the same as those of that disease. When it develops as the result of injuries to the head, the symptoms are also the same as in chronic meningitis.

OTHER CASES OF HYDROCEPHALUS.—Besides the congenital and secondary forms of hydrocephalus, we have cases in which the dilatation of the ventricles occurs during adult life, and apparently, in many instances, as a primary disease. After death an examination will show that the lesion is confined to either one or both of the lateral ventricles, and consists in their being dilated and containing an increased amount of clear serum. The ependyma lining these is thicker than normal, and the inner surface of the ventricles is at times roughened. The degree of distention varies in different cases, but is never so great as is found in congenital cases, and in some it is very moderate indeed. I have seen latterly two cases of this disease in which the symptoms were very well marked, and after death the increase in the size of the ventricles was so slight that I hesitated in assigning them to this class of cases. The ependyma presented the accustomed appearance, but the distention was comparatively moderate, so that I hardly know what degree of dilatation is necessary to prove fatal.

Etiology.—As to the cause of this condition we are ignorant. It is said that it is due to an acute or chronic hyperæmia of the brain, but of course this is merely a theory. It is supposed to be secondary to excessive bodily or mental exertion, but such explanations of its causation are not satisfactory. Some think that it is the result of obstruction of the veins. It is said to be due to the obstruction of the passage leading from the third ventricle into the fourth. This condition has been observed after death, but in the larger number of cases we find no satisfactory cause.

Occurrence.—As a rule, this disease is found in persons over thirty years old; usually between the ages of forty and fifty. It seems to occur among all classes of society, apparently having nothing to do with starvation.

Symptoms.—The symptoms are well defined in the sense of being pronounced, but not in the sense of being characteristic. We can, however, divide these cases somewhat artificially into two sets as regards symptoms.

In one set of cases the symptoms seem to resemble those of cerebral apoplexia. I will give you briefly the history of a man who died from this disease within a few months past. He was twenty-six years of age, a roofer, and had been in the habit of carrying slates upon the top of his head. It was this, in his opinion, that caused his trouble. He was apparently well until within seven weeks of his death. I may say that in these cases the period at which the symptoms develop is usually only a few weeks before the death of the patient. We have to suppose, I think, that the lesion is of slower development. In this particular case the first symptom which manifested itself was a pain in the head and a feeling of lightness. I may remark that this feeling is also sometimes described as a dizziness; at times the patients can hardly tell you what the feeling is like, except that they cannot work or apply their minds in any way. The pain and lightness in the head, however, were not persistent; there were some days when he was able to work; again these symptoms would return for a short time, and, passing off, on the next day he would resume work. Pain, accompanied by this peculiar feeling in the head, characterizes a moderately large number of these cases. With other patients the first symptom, instead of being pain, is impairment of the intellectual faculties. They begin to lose their memory, and are unnaturally

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drowsy or stupid. In other words, these cases begin very much as do those of chronic meningitis. The particular patient of whom I am speaking continued for a couple of weeks in the condition I have described, when the pain became more severe and persistent, so that he was obliged to leave off work altogether. Now, the pains, which were very severe, instead of being confined to the head, extended to the occiput, cheeks, and jaws. He also complained of having noises in his ears. This case is the only one in which I have noticed such a shifting of the pains. These symptoms continued until a week before he died, when they became very much more severe. His mental condition also finally changed. The man seemed almost idiotic; he was dull, stupid, and could not answer questions intelligently. Besides this he developed twitchings of the muscles of the face. He also had internal strabismus, and in this condition he remained until finally, when sitting up in bed, he suddenly fell back dead.

The history of this patient will answer as a picture of these cases. You will observe that the symptoms begin some little time before the patient's death; but at first they are not very marked. Within a short time of the patient's death the symptoms become more pronounced, and finally the patient lapses into a condition of idiocy, and, as a rule, dies suddenly. At the autopsy the only lesion observable is the distention of the ventricles, and possibly, roughening of the ependyma.

In other cases the symptoms are like those of apoplexy. Here, again, you have mild and indifferent symptoms beginning from two to five weeks before the patient's death. The first change noticed is usually in the intellectual faculties; there is failing of the memory, and the temper of the patient is changed. The man is not the same that he was, and yet there is nothing very positive in this change; you hardly know whether there is any disease developing or not.

Then there are patients in whom the symptoms are a little more marked than this. There will be temporary paralysis of one arm, or of the muscles of the face, and when this passes off, nothing will remain except a slight disturbance of the intellectual faculties. Thus the case will continue, until finally the patient will suddenly become completely unconscious and comatose, as if suffering from the lodgment of a large clot in one of the arteries of the brain. He may remain comatose for a few hours, or a few days, and die.

At the post-mortem, instead of having a clot or extravasation of blood, you will simply find distention of the lateral ventricles. In some of the older English works upon the diseases of the brain, you will find these cases described, and they are still described, as *serous apoplexy*, because of their resemblance to cerebral apoplexia. Although the lesion present in this disease is fatal, it is difficult to understand why it is so, for it seems comparatively slight.

TUMORS OF THE BRAIN.

We come now to the consideration of another lesion of the brain, namely, that of tumors. The subject of tumors of the brain is such an extensive one that I shall not attempt to go into it at all deeply. But there are some facts in relation to tumors of the brain that can be told you in a short time, and which will be of service to you.

These tumors, it matters not what their structure may be—whether benign or malignant—hard or soft, all produce the same symptoms; so that you do not need, from a clinical point of view, to trouble yourselves with their particular structure. It is important to remember that some of these tumors are syphilitic. It is also important to bear in mind, that these cerebral tumors can be developed either from the cranial bones, the membranes of the brain, or from the substance of the brain.

Symptoms.—A patient may have a tumor of the brain, and during life manifest no symptoms whatever of its existence, it being discovered only at the autopsy. Of the cases which do have symptoms, they are divided into two sets: first, those having general cerebral symptoms, and second, those manifesting local cerebral symptoms. The general cerebral symptoms simply depend upon the unnatural condition of the brain. Of these the most characteristic, are constant and severe pain in the head, and certain disturbances of the intellectual faculties; the patients are stupid, dull, and at times fairly idiotic. They may also have general convulsions.

The local cerebral symptoms are dependent upon the particular part of the brain involved by the tumor. For example, if the tumor is situated in the motor track of the brain so as to involve the two central convolutions, you will have upon the opposite side of the body either convulsive movements or paralysis of the muscles; at times both.

If the tumor involves the island of Reil, or the posterior portion of the third convolution, the patient will become aphasic. If it is so situated at the base of the brain as to press upon the optic nerve, the eyesight is lost. If it presses upon the auditory nerve, there is loss of hearing, either upon one side or both, according to the situation of the tumor. If the tumor is situated in the corpus striatum, and sufficiently large to involve the internal capsule, then you will have upon the opposite side of the body well-marked loss of motion, and with this, loss of sensation. So you may have different symptoms developed according to the exact situation of the tumor in the brain.

It is noticeable, however, that although we have general and local cerebral symptoms, they are not persistent in the majority of cases. The lesion persists, but the symptoms intermit. It is difficult to understand why this is so, but it is very commonly observed. Headache, change in the mental faculties, paralysis of certain groups of muscles, affections of the different nerves; all these will be sometimes present, sometimes absent. As the disease progresses, however, and the tumor continues to grow, these symptoms will become more and more persistent ; the intervals between the attacks will be less and less noticeable, and finally the patient will be confined to the bed, where he will remain in a quiet, drowsy, semi-idiotic condition, until he dies from an attack of convulsions, or suddenly, from reasons with which we are unacquainted.

Duration of the Existence of Brain-Tumors.—The length of time during which tumors exist varies according to their structure.

If the tumor be malignant, the patient will not live as long as he would if it were benign ; but this seems due to the fact that malignant tumors grow a great deal more rapidly than do benign tumors. It is surprising to know how large a tumor may be, and still the patient continue to live ; they may grow to be as large as an orange before causing death. The duration of the existence of benign tumors may be for many years, still the tumors steadily grow, and during this time the symptoms will be intermittent.

There are some cases in which the tumor exists apparently before the symptoms manifest themselves. These cases are very difficult indeed to diagnose. Such a patient will be brought into the hospital, or he may come under your observation, whose friends will tell you that he has always been perfectly well, and has had no cerebral symptoms whatever ; but that he was suddenly seized with an attack of convulsions, which was followed by stupidity or idiocy. In this condition he will remain until he dies, which may not be more than one or two weeks. At the post-mortem you will find a cerebral tumor of such size and structure that it seems evident that it has existed for a considerable length of time.

Recapitulation.—You will observe that we have these three sets of cerebral tumors, so far as symptoms are concerned. In the first set of cases there are no symptoms whatever, and we only discover the lesion after the patient's death, which is caused by some other disease. In the second set of cases the symptoms develop gradually ; they are intermittent, but extend over a number of years, and vary according to the position of the tumor in the brain. In the third set of cases the tumors apparently exist for a considerable time before any symptoms are developed. The symptoms come on suddenly, and the patient lives but a short time after their development.

LECTURE XIII.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Character, Pathology, Etiology, Invasion, Symptoms, and Course of the Disease.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.—We begin to day the study of epidemic cerebro-spinal meningitis. This disease belongs to the class of general diseases, and there is an impropriety in considering it in connection with diseases of the brain and spinal cord; but there are so few of the general diseases that fall to my share of the course, that it is more convenient to consider it now than later. You will remember, however, that it is something quite different from the diseases of which we have lately been speaking. It is a general disease, and its characteristic lesion is situated in the membranes of the brain and spinal cord, but it is not a local trouble of these parts.

Pathology.—In persons dead of cerebro-spinal meningitis, the characteristic lesion is found in the membranes of the brain and spinal cord. The piamater of the brain and spinal cord is infiltrated with pus, fibrin, and serum. In a few cases we also find pus and fibrin lining the inner surface of the duramater. The inflammatory lesion is often found over both the convexity and the base of the brain, and also over parts of the spinal cord, both upon the anterior and posterior surfaces, although, owing to the recumbent position of the patient in bed, pus, fibrin, and serum are more apt to collect over the posterior surface. In the ventricles of the brain there are to be found a number of pus-cells suspended in serum. These are all the lesions of epidemic cerebro-spinal meningitis.

There are some cases in which the disease will continue a long time, the patients living for weeks or months. Under such circumstances the ventricles are filled with turbid serum, the lesion being much more marked than in the acute cases.

In the bodies of some who die of this disease it is not possible to determine the existence of any lesion with the naked eye. This condition is observed during the prevalence of malignant epidemics, and in patients who die of the malady within a few hours. Under such circumstances, apparently, there is not time enough for the lesion to develop, and the patients die from the general poisoning of the disease.

The disease has been known since the beginning of this century. It has occurred in epidemics, and these have varied a great deal in their extent and fatality. Having occurred as an epidemic for one, two, or three years, in a city or town of some size, it is apt to remain as an endemic disease for many years afterward, a small number of cases occurring each year. Climate seems to have very little effect upon the disease, except that it has not been observed in the tropics.

It may occur at any season of the year, but is more apt to prevail during winter. It affects individuals at any period of life, from early childhood to old age, but it is more frequent in children than in older persons ; there have been epidemics in which the disease was confined to children. It occurs more frequently among people who are crowded together, as in large schools, workhouses, barracks, or prisons, but still it is not confined to these. Occasionally, however, epidemics are limited to the inmates of a single large workhouse, or to the children of one school. Many times whole towns, districts, or countries become the seat of the disease, and when this happens, persons will be taken sick in isolated dwellings. In New York the disease was epidemic in 1872 and 1873. It was not severe, or extensive, but still there were a good many cases at that time. Since then it has been endemic, and there have been a moderate number of cases every year.

Etiology.—We have no exact, or even probable, knowledge as to the local cause of the disease. We cannot say whether it is due to any conditions of the soil, or the result of decomposition, or whether its origin is connected in any way with sewer-gas. We do know, however, that the disease is not contagious in any sense of the word. Patients do not need to be isolated any more than do those with pneumonia.

Invasion of the Disease.—Patients attacked during the prevalence of epidemics, in which the disease occurs in its most fatal and malignant form, only live a few hours, or days. The invasion of the disease is exceedingly abrupt. A patient who has been apparently in perfect health will be suddenly seized with a chill, lose consciousness, become comatose, and may die in a few hours. If he lives a little longer than this, there may be time for a febrile movement to be developed.

Sometimes patients while walking along the street will be unexpectedly attacked by the disease, fall to the ground unconscious, become comatose, and by the time they have been carried to their homes, will be dead. There is scarcely any disease which can assume a form so rapidly fatal as this under certain circumstances. Epidemics of malignant cerebro-spinal meningitis are fortunately quite rare. There have been none in this country, and only a few in other countries.

In ordinary epidemics there is considerable difference as to the invasion of the disease, its course, and fatality. In some a considerable number of patients die, but not in the very rapid way of which I have spoken. There are other epidemics in which most of the patients get well ; and so, between these two extremes, there are many grades in the fatality of the disease.

As the malady has been observed in Germany, during the past few years, its invasion has been sudden. The patients have been attacked by chills, rapidly followed by a febrile movement, and have at once developed a severe headache, usually accompanied by vomiting. They have rapidly developed delirium, and within the first twenty-four hours have become quite sick.

In England, on the other hand, most of the cases have not been attacked suddenly. There has been a period preceding the marked invasion of the disease during which the patients have suffered from a general indisposition. They may have a diffuse headache, neuralgic pains in the back and different parts of the body, loss of appetite, a slight irregular feeling of chilliness, and a little febrile movement.

In this condition of indisposition they may remain for days or weeks and then develop well-defined symptoms.

In New York, considerable variety has been observed as to the beginning of the disease. Some patients in ordinary health have been suddenly attacked by well-defined rigors, which were very soon followed by a pronounced febrile movement. They have at once complained of severe headache, usually frontal; there have been some disturbances of the stomach, and a little vomiting coming on after a few hours. On the second day they have had a temperature of 99° or 100°, and the patients have been, by this time, fairly under the influence of the disease.

In other cases the invasion has not been quite as abrupt. The patients have, at first, complained of pains in the back of the neck, in the head, and all over the body. They would have no rigors, but after the lapse of twenty-four or forty-eight hours a febrile movement would be developed, and the patients would then pass fairly under the influence of the disease. In still other cases the invasion has at once been marked by severe headache and persistent vomiting, there being no rigors,—no period of prodroma. In a few hours the patients have become delirious, and shortly after a febrile movement has developed, following which they have had the ordinary symptoms of the disease. In other cases the invasion has been exceedingly slow. The patients have complained of general muscular pains of a moderate degree, and pain in the head. Perhaps the head would be turned back a little, owing to the pain in, and partial contraction of the muscles of the neck and back. There would be no marked symptoms of the disease for four or five days. In some cases there is conjunctivitis, and, curiously enough, this may be the symptom for which you will be consulted.

I had a patient last winter who came to me for sore eyes. Upon inquiry I found that for several days he had been suffering from headache, and irregular muscular pains all over the body. He had been unable to follow his business, but had no idea that he was about to have a severe disease. The next day the disease was fairly developed, and went on to run its regular course. These are the different ways in which the invasion of the disease is marked.

In New York, the cases in which there has been a sudden invasion of the disease were more common, while the malady was prevailing as an epidemic; now that it is endemic, we see more cases in which there is a period of prodroma, during which there are irregular and indefinite symptoms.

Symptoms and Course of the Disease.—When the disease is fairly developed there are certain regular symptoms. In the first place headache is very constant. I do not know that there are any cases in which it is entirely absent; but its severity varies considerably in different patients; in some it is exceedingly severe, in others quite mild. It may be a localized frontal one, the patients saying that the rest of the head does not pain them. Occasionally it will be confined to the occipital region; and in still other cases it will be a general headache. Some patients will constantly complain of pain in the head, and even after they are delirious it will be evident that they still suffer from it. In others it will be mild, and if you ask them in regard to it, they will say that their head aches, but they will not complain of this. In some cases the headache is continuous; in others, there will be remissions, and the patient will be free of it for a few hours; then it will return.

The larger number of patients become delirious. This symptom, however, is not as constant as the headache. In the mild cases the delirium may be entirely absent. In those that are a little more severe, the patients are only delirious at intervals, especially during the night. In the worst cases the patients are delirious all the time.

Delirium may be present at the very beginning of the disease, or it may not be developed until the patient has been sick for several days. Generally speaking, if it comes on early, it indicates that the case will be a severe one, and the prognosis is not as good as when it is developed later. The character of the delirium also varies in different cases. Some patients will think they see people whom they do not see. If left alone they will be disposed to get out of bed, and go out of the house; but they are easily retained. In other patients it is very severe and they can hardly be held in bed; sometimes they require to be tied in bed. Between these two extremes there are many variations as to the violence of the delirium.

In the bad cases the intellect will be affected early in the disease; patients will at once, or in a very short time, cease to take notice of what is going on about them. In the mild cases the intellectual faculties will continue tolerably good during the entire disease, the patient being able to answer questions intelligently. Sometimes they can transact business, although they do this with great difficulty. There are other cases in which the mind is not affected until after the first week of the disease. The temper of the patients changes. They are almost always very peevish and irritable, though their minds are still unclouded.

In adults general convulsions occur only in a moderate number of cases; in children, however, they are very common, especially during the invasion of the disease, or during the first twenty-four or forty-eight hours of its existence.

Vomiting is a very common symptom at the beginning of the disease, and it may be the most marked symptom. Patients will indeed also have headache and pains in other parts of the body, but the vomiting will be so frequent and distressing as to mask the disease. Under such circumstances it is possible to mistake it for acute gastritis ; more frequently, however, the vomiting does not excite so much attention, and after the first few days of the disease the stomach becomes quiet.

The bowels are usually constipated, not obstinately, however, for they can readily be moved by the use of medicine. It is possible to have a diarrhœa at any period of the disease, but this is more common near its close in bad cases.

Besides pains in the head, patients are very apt to have pain and twitchings in some of the voluntary muscles, especially in those of the back of the neck and along the vertebral column.

In some cases there will be pain and tenderness upon pressure, and this will involve nearly all of the muscles throughout the body. With this pain there will be unnatural contraction of the muscles, in consequence of which the head and the whole body will be turned backward. Occasionally there is hyperæsthesia of the skin, and patients may even complain of the pressure of the bed-clothes.

The ears are very apt to be affected, the patients becoming deaf during the course of the disease, and, in some cases, remaining so after its termination.

In a large number of patients, especially in adults, there is inflammation of the conjunctiva, and this may occur, as I have said, early in the disease. In some cases there will be, in addition, inflammation of the cornea, which is very apt to be of a suppurative character, so that portions of the cornea are destroyed and the eyesight permanently injured. Some patients may have a suppurative inflammation of the choroid, and when this happens, the eye is always rendered useless for vision. Such inflammation may involve one or both eyes. Besides inflammation there are also other changes to be noticed in the eyes. The pupils may be dilated or contracted ; in either case not responding to light. There is quite often internal strabismus and a certain degree of photophobia, which seem to depend upon the conjunctivitis and choroiditis.

In a certain number of cases there will be marked eruptions of the skin. It may have the characteristic appearance of erythema, or roseola, or of a peticular eruption. Such eruptions may be present in some epidemics, and altogether absent in others. Epidemics in which the eruption is marked, are sometimes known under the name of *spotted fever*.

The tongue at the beginning of the disease is moist and coated with white fur, but after the disease has lasted for some time it gradually becomes dried, and the white coat becomes brown, looking like a tongue of typhoid or typhus fevers.

The pulse is generally full and strong, and not very rapid; it does not usually exceed a hundred beats to the minute, and very often not more than fifty or sixty. If, however, the case is to prove fatal, during the last days of the disease the character of the pulse changes, and instead of being full and rather slow, it becomes very rapid. When I speak of a slow pulse, in this connection, I mean a slow pulse for a patient suffering from a febrile movement. The temperature in this disease is irregular and does not run a characteristic course. In many cases there is at no time a temperature exceeding 102°, 102.5°, or 103°. There may be days during which it will be between 99° and 100°, but in the more severe cases it may run up to 104° or 105°. In children the temperature runs higher than in adults. The height of the temperature is not indicative of the course of the disease; you cannot judge how a patient is doing by the height of the temperature. The things to be remembered in regard to the temperature are: that it is irregular, that it is higher in children than in adults; if it is high throughout the whole of the disease the prognosis is, as a rule, worse than when it is more moderate; but whether it is high or low you cannot base a prognosis upon it.

Upon the chart before you I give the temperature from the second to the eighteenth day of a patient who died of cerebro-spinal meningitis. Upon the second day the temperature in the morning was 99.5°; in the evening 101.5°; the next morning 101.5°; the third day it ran up to 103°; the fourth day it went down to 102° in the morning, but in the evening was 105°; on the fifth day the temperature ran up in the morning and down in the evening; the same was the case on the sixth day; in the morning and evening it was 99.5°; on the following morning it was 103.5°; and so you will observe the temperature running up and down in this irregular way until the fifteenth day of the disease, when it reached 106°; after reaching this point it fell again to 102.5°, when the patient died. Now all this time the patient was really getting worse; there was no abatement of the disease at any time, and yet during a period of seventeen days the temperature ran an irregular course; on the morning of the thirteenth day it ran down to 100°; and on the following morning it was 105.5°. The pulse, you will observe, as shown by this chart, also varied as much as the temperature; but most of the time it was comparatively low, not over ninety-five, until the last few days of the disease. Then it began to run up, and upon the day of death it was one hundred and fifty; during the early part of the disease the pulse was eighty, sixty, and one day went down to fifty.

The combination of a tolerably high temperature, as 103° or 104°, with a pulse of sixty or seventy, is always very good reason for thinking that the patient is suffering from meningitis; but the particular kind is to be determined by other observations.

If the disease lasts more than ten days, or more than two weeks, the patients are very apt to pass into a typhoid condition; the tongue becomes brown and dry and the delirium is constant. The patients lie in bed quietly, except for the disturbances produced by urination. They may pass their urine and fæces involuntarily; their cheeks may be flushed so as to present the appearance of patients suffering from typhoid fever. This condition is not usually developed until after the patients have been sick for ten days or two weeks.

The duration of the disease in very malignant cases is only a few hours; in the more ordinary cases it varies between a week and three weeks. The more severe the case is the shorter its duration. Of the cases which prove fatal, a very considerable number die at the end of the first or second week; of the cases which get well, the disease is apt to be protracted for two or three weeks; in the cases which are to get well, you sometimes have a sort of relapse, not as well-marked as in typhoid fever, but still easily recognized. It occurs somewhat in this way. At the end of about two weeks the patient may be apparently quite well; the delirium will have disappeared; the temperature will be normal, the intelligence clear, and the appetite good; after continuing in this way for two or three days, the temperature will run up, the delirium will return, there will be loss of appetite, and the patient will be in every way in much the same condition as before; there may be several of these moderate relapses, after which convalescence will finally be established.

LECTURE XIV.

EPIDEMIC CEREBRO-SPINAL MENINGITIS—CONCLUDED.—DISEASES OF THE SPINAL CORD.

Epidemic Cerebro-Spinal Meningitis; Complications, Sequelæ, Prognosis, and Treatment.—Diseases of the Spinal Cord.—Anatomy and Physiology of the Spinal Cord.—General Symptoms of Diseases of the Spinal Cord.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.—*Complications.—Gentlemen:* To-day we will continue the study of epidemic cerebro-spinal meningitis by considering first its complications.

Bronchitis occurs as a complication in some cases; pneumonia, though less frequently, in others; pleurisy, pericarditis, and endocarditis are also met with as complications, the latter affection leaving, in some cases, a permanent lesion of the valves.

Sequelæ.—The disease is also attended with certain sequelæ. Blindness may continue after the recovery of the patient, if due to destructive changes. A certain number of patients do not get well, but go on to suffer from a form of chronic hydrocephalus, the ventricles of the brain being distended with serum and the patients having the symptoms of chronic hydrocephalus. Some patients recover with impairment of the mental faculties; or occasionally there may be loss of speech. The feebleness of mind and aphasia, fortunately, are not always permanent; they may continue for some months after the recovery of the patient, and then gradually disappear. I have at the present time under my care a young girl in whom this condition was very well marked. She suffered in March last from cerebro spinal meningitis. After her recovery she was left completely aphasic, and very feeble-minded; this condition lasted until June, when it gradually began to disappear, the aphasia, first, followed by slow improvement in the mental faculties. She continued to improve during the summer and fall, and now she seems to be as well as ever. This same case presented another complication which has persisted. She suffered from endocarditis at the time she had cerebro-spinal meningitis, which has resulted in a permanent lesion of the aortic valves—a lesion which did not exist before her sickness.

You should remember, then, that the prognosis in regard to the aphasia and the mental condition is not very bad. In some cases there remains paralysis of some of the voluntary muscles; there may be perhaps paralysis of an arm or a leg, or of a group of muscles in one of the extremities. This is most always permanent; or, it may last for a longer or shorter period, after which the patient will gradually regain power over these muscles.

Prognosis.—The prognosis of the disease varies very much with the particular epidemic which is prevailing. There are some epidemics in which it is bad, the larger number of patients dying; there are others in which it is very much better, and in which the larger number of patients get well. Generally speaking when the epidemic has subsided the disease runs a comparatively mild course. This is true of the greater number of cases now occurring in New York; the patients get well after the disease has run a mild course.

Treatment.—In the treatment of this disease you have to remember that it is a general disease attended with a local inflammation, and that, as a result of this, you have patients suffering from symptoms depending upon two distinct causes: one of these being the general poisoning of the patient; the other the local inflammation which accompanies the disease. Our treatment of the general poisoning of the patient is not of any particular efficacy, for we do not know how to act upon the poison of cerebro-spinal meningitis any more than do we understand how to act upon the poison of typhoid fever, small-pox, or any of that class of diseases. For the local lesion, however, we can do something, and for the symptoms produced both by the general disease and local lesion we can also do something. The indications for the treatment of the local lesion are the same as in acute meningitis of the traumatic or tubercular varieties; that is, at the very commencement of the disease local bloodletting and the application of cold may be resorted to. Local bloodletting is never employed in persons who are not strong and robust, and blood is to be taken from the temples, nape of the neck, and from the skin over the upper part of the spine by means of leeches and wet cups. This, however, should only be done during the first three or four days of the disease, as after this time there could be no object in resorting to this measure. Cold is to be applied pretty constantly during the first week of the disease to the head and back of the neck, by means of ice-bags. In a considerable number of cases these two means have been proven to be the most efficient agents we have for controlling the local inflammation; but they are only efficient in the early part of the disease. Bloodletting during the first two or three days, the application of cold during the first week of the disease. In addition to these local measures we have others intended to make the patients more comfortable. The symptoms which usually trouble patients most are headache, restlessness, and delirium. The use of bromide of potassium, given alone in twenty or thirty-grain doses every three hours, or in combination with chloral hydrate or hyoscyamus, or one of the preparations of musk, will most commonly be found the best means of quieting these patients. The bromide can be used in almost all cases, but it will be even more efficient by the addition of other drugs, one of the best being chloral hydrate; there are, however, some patients who cannot take chloral. With such the tincture of hyoscyamus will be found very efficacious, given in drachm doses with the bromide. Occasionally you will be obliged to resort to opium, for neither the bromide nor other drugs will answer the purpose; if possible, however, you should avoid its use. Quinine is not indicated in this disease, and there is no practical advantage in giving it, unless you do so as a matter of routine.

The temperature, I think, does not require any particular attention, for it does not run very high except toward the close of the disease, in fatal cases, when there is no efficacy in any treatment. In adults, a temperature of 104° is, I think, very harmless, and it may last for a considerable length of time, without any injury to the patient whatever. If, however, you are disposed to lower the temperature, the best way to do this is by means of cold effusions, or cold or tepid baths. Quinine has no effect whatever in reducing the temperature in this particular disease.

Occurrence in Children.—In children cerebro-spinal meningitis runs a little different course from what it does in adults; the diagnosis is by no means as easy, especially if the child is quite young. The invasion of the disease is, as a rule, acute, the patients becoming sick at once; it is very apt to be marked by convulsions, the patients having a number of these, repeated at intervals during the first twelve, twenty-four, or forty-eight hours. The convulsions then disappear, but the children continue dull, stupid, and drowsy; you can hardly say they are comatose, but they take very little notice of what is going on around them. The temperature runs higher in children, from the commencement of the disease, than in adults, not infrequently reaching 104° within the first twenty-four hours, and afterward continuing at from 104° to 106° or 107°. In children these may be the only symptoms, the patients continuing in this stupid condition with a well-marked febrile movement until they die; or, after a time the stupor may gradually disappear, the temperature become reduced, and the patient get better. The disease is much more fatal in children than in adults. In a considerable number of cases occurring in children you will find it impossible to tell whether the patients are suffering from epidemic cerebro-spinal meningitis, acute tubercular meningitis, acute meningitis, or suppurative inflammation of the middle ear, for the symptoms of these various conditions are about the same.

Treatment.—The treatment is the same as in adults, only in these young children local bloodletting is never resorted to.

DISEASES OF THE SPINAL CORD.

Anatomy.—We come now to the consideration of the diseases of the spinal cord.

If we make a transverse section of the spinal cord, we see at once that it consists of two parts. One of these we call the gray matter, the other the white matter of the cord.

The gray matter is arranged in what are called horns or cornua, and we speak of these as the two anterior horns or cornua of the gray matter, and the two posterior horns or cornua of the gray matter. The white matter is arranged in what are called columns, which run the whole length of the spinal cord, and these, according to their position, are spoken of as anterior, posterior, or lateral columns. These are all the divisions of the cord which it is practically necessary for us to remember. As you already know, the anterior nerve-roots connect with the anterior gray cornua, and the posterior nerve-roots with the posterior gray cornua.

Physiology.—There are some points in the physiology of the spinal cord which are necessary to be borne in mind when we come to the consideration of diseases of the cord. We know that impressions received by the sensitive nerves of the body (peripheral) are carried to the posterior roots, from thence to the posterior gray cornua, and from these through the posterior and lateral white columns to the brain; so you will remember that the posterior and lateral white columns are channels by which sensations, which we designate by the terms touch, pressure, and temperature, are conveyed from the periphery of the body to the brain. The sensation of pain is distinguished from these, and is probably transmitted through the gray matter of the cord; of this we are not absolutely certain, but it is exceedingly probable that such is the case. We can distinguish these two channels through which sensations are conveyed from the periphery of the body to the brain. The lateral and perhaps the anterior columns of the cord transmit the voluntary impulses to the anterior cornua; and the anterior cornua of the gray matter have the power of regulating the impulses transmitted through the brain to the voluntary muscles.

The centres of co-ordination for different voluntary muscles are probably situated in the brain, and the impulses from these centres are transmitted through the posterior white columns. Reflex motion, as it is called, in contradistinction to voluntary motion, is produced by the gray matter of the cord ; but these are normally controlled by the influence of the brain, so that if the influence of the brain is taken away, these reflex motions which belong to the spinal cord will no longer be controlled, and will become exaggerated. These are points which it is necessary for us to remember in thinking of diseases of the spinal cord.

General Symptoms of Diseases of the Spinal Cord.—There are some general symptoms belonging to a number of different diseases of the spinal cord which we also ought to remember. We know, in the first place, if a person has any disturbance of sensation as to heat, cold, or pressure, or, if there is any disturbance of tactile sensation, or feeling of pain, there must be a lesion either of the gray matter or posterior white columns of the cord, or both. Subjective sensations, such as feelings of heat and cold, or numbness of the skin, or pricking, or formication in the skin ; with all these unnatural feelings we are still uncertain as to what part of the spinal cord they are to be referred, or whether they are to be referred to any part of the spinal cord. If patients suffer from well-marked neuralgic pains these are due, so far as we know, to changes in the posterior nerve-roots, which are given off from the posterior gray cornua. Our information upon this subject is not exact, we can only say, so far as we know, for we have no actual knowledge as to whether it be due to changes in the posterior nerve-roots or to changes in the cord itself. The general pains in different parts of the body belong to the class of uncertain symptoms. We do not know what particular part of the cord it is which gives rise to these. Paralysis of the voluntary muscles, when rapidly followed by atrophy, is due to disease of the anterior horns of the gray matter, or the anterior nerve-roots given off from these, or both of these. If there is paralysis of the voluntary muscle accompanied with permanent contractions, and there is no atrophy, we can usually infer that the lateral white columns are diseased. If patients suffer from tremor of the voluntary muscles, from the peculiar agitating movement which we see in certain cases, or paralysis agitans, as it is sometimes called, it is due, as a rule, to what is called multiple sclerosis ; that is, there are a number of foci of the lesion in the spinal cord, and there may be, at the same time, similar foci of the lesion in the brain. If patients suffer from diminished reflex action, this is commonly due to disease of the anterior or posterior nerve-roots, or to disease of the gray matter of the cord. If there is exaggerated reflex action, this is due to some lesion which interrupts the natural channels of connection from the brain to the spinal cord. If a patient falls from a height and dislocates one of the cervical or upper dorsal vertebra, so as to compress the cord, you will find that he will have exaggerated reflexes of the parts of the body below the lesion. This condition is seen in many of the transverse lesions of the spinal cord.

LECTURE XV.

DISEASES OF THE SPINAL CORD—CONTINUED.

Congestion of the Spinal Cord; Etiology, Invasion, Symptoms, Prognosis, Diagnosis, and Treatment.—Myelitis; Pathology, Etiology, Invasion, Symptoms, and Course of the Disease; Complications and Treatment.

GENTLEMEN: We were speaking last time of some of the general symptoms belonging to diseases of the spinal cord.

CONGESTION OF THE SPINAL CORD.—The first disease of the cord to which we will direct our attention is commonly described under the name of *congestion of the spinal cord.* Cases described under this arbitrary name have certain definite clinical features in common, and hence have been grouped together and spoken of as constituting a special condition of the cord. But we do not really know that any such congestion exists; it is only assumed. This is a disease which is not fatal, so that no autopsies have been made. Even if these patients should die, we know very well that, from an examination after death, we cannot form an accurate judgment regarding the condition of any viscus that has been congested during life; and when we speak of these cases under this name, we do so because it is general practice.

Etiology.—We are also ignorant of their causation. In some cases the disease comes on after exposure to cold; in others after venereal excesses; again it occurs after accidents, in the case of persons who have been very much shaken up, or have fallen from a height, or have been injured by a railroad accident. Besides these there are other cases in which no cause can be assigned.

Invasion and Symptoms.—The invasion of this disease is, as a rule, sudden, a patient may go to bed in perfectly good health, and awake during the night with the symptoms of the disease. Patients complain very often of pain in the small of the back; but this does not differ from the pain in this locality which is complained of under many other circumstances. It is, however, a tolerably constant symptom. There may be disturbances of sensation, although these are not constant. Patients may complain of a feeling of numbness, either in the arms or legs, or in both; or they may experience a feeling as if ants were crawling over the skin, or as if pins or needles were being thrust into the skin; or there may be some loss of tactile sensation. There is always paralysis of the voluntary muscles, usually of the legs, which is, however, at first not complete; the patients, although not having full power over the muscles, are still able to move them. It may remain confined to these, or it may extend rapidly upward and involve all of the voluntary muscles below the neck, all these becoming partially paralyzed. There may also be convulsive twitchings of the voluntary muscles, but this is not a constant symptom, and when it exists, it usually does not reach any very great degree of severity; nothing like general convulsions. The bladder is very often affected, patients having retention or incontinence of urine. There may be constipation, or the fæces may escape involuntarily, the former condition, however, being the more common. These patients very seldom develop bed-sores, although it is possible for them to do so.

These symptoms come on very rapidly. A patient may go to bed feeling in perfect health, and the next morning, without feeling sick or having any idea that there is anything the matter with him, will try to get up, when he will fall to the floor because the muscles of his legs are not strong enough to hold his body. He will return to bed, and it will be found that he can move his legs, but still they are not strong, being in a condition of partial paralysis. The physician, on being sent for, comes and finds the patient without any febrile symptoms, but with simply loss of power in his legs, and in addition he may find that the feet or toes of the patient are numb, or that he complains of a peculiar prickling sensation or pain, and that he cannot pass his water. In this condition the patient may remain for a few hours, and then the loss of power in the voluntary muscles may begin to extend upward, and in the course of two days the patient may be completely paralyzed below the neck. The urine will be either retained or dribble away. There will be constipation or incontinence of fæces, and the patient will not be able to help himself at all. He may move his legs and arms a little, but not enough to help himself. In other cases, however, there will be no such extension of the paralysis. It will be confined to the legs, and the duration of such cases is usually shorter than those in which the paralysis extends to other voluntary muscles. If it remains confined to the legs, it may not continue more than a couple of weeks, and then disappear altogether. The body above may remain paralyzed for weeks or months, after which time power will gradually return.

Prognosis.—The prognosis of this disease is good. The paralysis gradually disappears, and the patient returns to health. Patients once having suffered from this condition are not necessarily exposed to second attacks: the attacks are usually single.

Diagnosis.—The diagnosis of this condition is, at first, usually not easy, but after a time becomes less difficult. The conditions with which we are likely to confound it are myelitis, inflammation of the anterior gray cornua of the spinal cord, and hysteria. If the disease occurs in females, at the commencement of the attack a diagnosis cannot be made in many cases. After the disease has lasted for a short time, and you find the paralysis, after reaching a certain point, does not advance; if you find the disturbances of sensation become less marked, or more marked; if you find no atrophy; if the patient continues in generally good condition; all these will help you to make a diagnosis. To differentiate between this condition and that of hysterical paralysis, which occurs in some young women, is very difficult indeed. This condition may last some weeks, and after the patients have recovered, you may be in doubt as to whether the case was one of hysteria, or whether it belonged to that set of cases which we include under the name of congestion of the cord.

Treatment.—The treatment of this condition consists in the use of cups applied along the course of the spinal cord, and the administration of purgatives and of ergot. Of these three remedies the purgatives are apparently most efficacious. It is not very easy, I think, to say why they are efficient. If purgatives are used, they should be active, and I do not know that there is any drug of this class more so than croton-oil, given in doses of from one to three drops according to the strength and age of the patient. This will cause a number of loose movements of the bowels. Its administration should not, however, be continued for any length of time. After giving it the first time you should wait a few days, and then purge the patient again. This plan of treatment is usually the most efficient.

Next in efficiency seems to be the use of cups along the spinal column, and dry cups will answer the purpose as well as wet ones. They should be used very thoroughly, and when applied properly, they not only draw the blood to the skin and subcutaneous tissue, but they will produce an extravasation of blood beneath the skin, this being, in reality, subcutaneous bloodletting. The blood remains extravasated until it is absorbed. The cups are to be applied thoroughly every day, or every second day for several days, so as to get a decided effect. Perhaps the least efficient treatment is the use of ergot. This is more applicable after the disease has lasted for several weeks. At the beginning the indications are to use purgatives and dry cups, but after the lapse of several weeks you may give large doses of ergot. The fluid extract should be used in drachm doses, three or four times a day, according to the condition of the patient. The success of the treatment is, of course, very much aided by the natural character of the disease which is to terminate in recovery. The good effects of treatment are in hastening the recovery, and the remedies I have spoken of seem to do this. There is one difference between these cases and those of hysteria. These three methods of treatment, although of use in congestion of the cord, are of no use in hysterical cases of paraplegia.

MYELITIS.—*Pathology.*—The next condition of the cord which we have to consider is myelitis, or inflammation of the spinal cord. When a patient dies with myelitis the gross appearances of the spinal cord vary with the extent of the lesion, or with the degree to which the lesion has been developed before the patient's death. If the lesion is a small one, there may be no gross changes in the appearance of the spinal cord. If, however, the lesion is more developed, a certain part of the spinal cord is found to be softer than normal. If you lay the cord out upon a table, and run your finger over it, you will find some points where the cord is evidently softer than it should be. These softened patches measure from a quarter of an inch to an inch. The degree of softening also varies, sometimes existing only to a slight extent. In other cases it is so marked that the cord will be flattened at this point, and in still other cases the cord will drop apart if held up. This extreme degree of softening may even lead to rupture of the cord during the life of the patient. Of course the occurrence of this is rare, but it has been observed in patients suffering from myelitis, who have sustained a sudden jar or motion of the cord. The lesion is transverse, and it is very apt to begin at that point in the spinal cord where the lateral columns and the middle point of gray matter are in contact. It usually begins in the gray matter and extends into the white matter. After a time, if the lesion continues, it will involve more and more of the cord until the whole of this particular part of the cord (that is, the cornua) is converted into soft granular matter. As a rule we do not find pus-cells, but we find that the nerves, fibres, and connective tissue are replaced by a peculiar granular matter which does not belong to the cord. In addition to this we have, in a certain number of cases, well-marked thickening of the walls of the blood-vessels. Although the lesion is transverse, it is not usually limited to its primary seat. If you examine the cord above and below the softened patch you will find lesions of the same character, but not developed to the same extent.

Etiology.—The causes of this condition of the spinal cord are unknown. We find it in young persons who have indulged in venereal excesses, in old cases of syphilis, in persons who have fallen from a considerable height, but in the larger number of cases that come under observation we are unable to find any cause for the disease, and, under such circumstances, it is spoken of as occurring idiopathically.

Invasion.—Symptoms and Course of the Disease.—The invasion of the disease is not the same in all cases. In a certain number of cases the onset is sudden and there are well-defined constitutional symptoms. The patients are attacked with vomiting, headache, a decided febrile movement, delirium, and loss of power in some of the voluntary muscles. All these symptoms are developed rapidly. A patient who has previously been in apparently good health, will suddenly begin to vomit and complain of a headache. His temperature will run up, and he will become delirious, while, at the same time, there will be loss of power in some of the voluntary muscles, usually in those of the leg. In other cases the invasion is marked by the sudden occurrence of paraplegia, but the patients do not have a febrile movement. There is no disturbance of the stomach, and no headache. The paralysis may develop so rapidly that, within twenty-four hours, the patient will be unable to walk or lift his legs.

In still other cases, and more frequently, the invasion of the disease is gradual. The patients do not have any decided constitutional symptoms, but the first symptom noticed is, perhaps, disturbance of sensation usually referred to the feet. The patients complain that they have a feeling of numbness in the toes; that they cannot feel the ground under their feet as distinctly as they should, or that they feel as if something was crawling over their feet or legs, or that pins were being brought into contact with the skin of the legs or feet. These disturbances of sensation may sometimes begin at the same time in the hands and feet. After this the patient begins to find that his legs have an unnatural feeling of stiffness and heaviness. He is still able to walk about, or, perhaps, is still able to work, but he finds, after walking or working, his legs are much more fatigued than commonly. This condition may last for several weeks. There may be, at first, no other symptoms. These are, however, gradually succeeded by more marked ones. The loss of power becomes more and more manifest, usually from day to day, although sometimes the change will be sudden. Finally, there is more or less complete loss of power in the legs or in the arms, which, of course, depends upon the particular part of the spinal cord in which the lesion is situated. If the lesion is high, the loss of power begins in the arms and legs; if lower, it begins in the legs and remains confined to them. After the loss of power in the voluntary muscles has lasted for a shorter or longer time, the patient has retention or incontinence of urine. In some patients this will be the first symptom, but it does not usually occur until later. Constipation commonly exists, and may be followed by the involuntary passage of the fæces.

In some cases loss of sensation comes on gradually, together with loss of muscular power. In other cases the loss of muscular power will be very noticeable before there is much change in sensation. This depends upon the particular way in which the lesion extends transversely; that is, whether it involves at once the parts of the cord which control motion and sensation, or whether it involves, first, those parts which control motion, and later, those that control sensation.

The disease is always a progressive one. The rapidity with which patients get worse is subject to great variations. Some decline rapidly, some slowly, but, sooner or later, there is complete loss of power and sensation in the legs, and absence of the reflexes. In this condition the urine and fæces are passed involuntarily. If the lesion is situated higher there is loss of power and sensation in the arms, and all the parts of the body below the seat of the lesion. In some instances patients will complain of a feeling of constriction about the body, commonly corresponding to the position of the lesion in the spinal cord. In this condition of complete loss of motion and sensation, and complete paralysis of the bladder and rectum, patients may continue to live for months and years.

Complications.—The lesion of the cord is not in itself fatal; patients do not, as a rule, die from myelitis, but from its complications. One of the most common complications is that of bed-sores, which consists in an inflammatory process being developed in those parts of the body which are in contact with the bed. The skin over the sacrum and hips is likely to be affected in this way, and to result in the death of the skin and tissue beneath. When once begun they are apt to constantly become larger and deeper, and in this way form a fatal lesion.

Next in frequency to this complication is the development of cystitis, or inflammation of the bladder, due to retention of urine. If the bladder is not emptied thoroughly the urine that remains in it is apt to decompose and give rise to an inflammatory process in the mucous membrane, with the production of mucus and pus. The cystitis in itself would not be so formidable a lesion if it were not that the patients so affected are liable to acquire suppurative inflammation of the kidneys, which is a fatal lesion, destroying life usually within a few days after its development. This is one of the ways in which a very considerable number of patients with myelitis die.

Another complication is pneumonia, and this is apt to be different from the ordinary lobar pneumonia of adults. These paraplegic patients may indeed suffer from lobar pneumonia, and die from it, as other patients; but there is a peculiar form of pneumonia from which they usually die. It is lobular pneumonia. The lungs become studded with little lobules of hepatization.

The above are the three ways in which patients affected with myelitis commonly die; but there are a few exceptions. Some patients, especially those in which there are acute constitutional symptoms at the beginning of the disease, seem to die, after a few days or weeks, from the myelitis.

Treatment.—There is not a great deal to be said in reference to treatment. I do not think it is wise to treat these patients very energetically, for so far as we know, when a patient has really developed a myelitis, the cord never returns to a normal condition, and the destruction goes on until the patient dies, or a considerable part of the cord is destroyed. I think we have no good reason to believe that any remedies which we possess can have any influence whatever upon this condition of the cord. It is always possible that we may be mistaken in our diagnosis, there being congestion of the cord instead of myelitis. If this is the case the patient will get well. The best thing that can be done is to endeavor to prolong the patient's life, and to keep him as comfortable as possible. In order to prolong his life you must endeavor to keep off complications. Bed-sores can be prevented by very great care. Never allow pressure to remain upon one part of the body for any great length of time. Having once formed, it is very difficult to get them to heal. The use of either the faradic or galvanic current seem to be of great service in helping the bed-sores to heal. The cystitis is to be treated by regularly emptying the bladder;

if you find the urine contains pus, in addition to drawing it off you must wash out the bladder. As regards pneumonia I do not know that there are any means of preventing its occurrence.

LECTURE XVI.

DISEASES OF THE SPINAL CORD — CONTINUED. — MYELITIS OF THE ANTERIOR HORNS.

Myelitis of the Anterior Horns; Pathology.—Occurrence of the Disease in Children.—Occurrence of the Disease in Adults; Acute and Chronic Invasion; Symptoms and General Course of the Disease.

MYELITIS OF THE ANTERIOR HORNS.—*Gentlemen:* The next disease of the spinal cord which we have to consider is known by several different names. It is called inflammation of the gray anterior cornua, also poliomyelitis anterior, and, when it occurs in children, infantile paralysis. When found in adults it has been named acute spinal paralysis, and in some cases, where it runs a chronic course, it is called chronic spinal paralysis. Perhaps as good a name as any is *myelitis of the anterior horns.*

Pathology.—The lesion involves, primarily, the anterior horns of the gray matter, and especially the nerve-cells situated in them. In the severer cases the lesion is not limited to the anterior horns, but extends backward into the posterior cornua, and latterly, so as to involve a part of the white columns of the cord. In children the disease is most marked at the lumbar and cervical enlargements of the cord; but it involves, to a certain extent, nearly the whole length of the cord—sometimes its entire length.

In a number of cases the patients recover entirely, and when this happens the cord apparently returns to a natural condition. The lesion in these cases is not a destructive one, the parts of the cord involved by it being simply changed for the time, and returning to a natural condition again. In other cases, when the disease is more severe, the parts of the cord involved do not return to their normal condition, and the lesion is destructive to some degree. The part of the cord which was diseased becomes permanently atrophied. There is a destruction of some of the nerve-elements of the cord, which are never replaced. Permanent atrophy of the cord is much more common in children than in adults.

Occurrence of the Disease in Children.—When the disease occurs in children it is most frequently found in those between the ages of one and four years, which is the period when the first teeth are being formed. It occurs with equal frequency in male and female children. In some cases the disease in children seems to follow prolonged exposure to cold; it also occasionally follows injuries, such as severe falls or blows. It occurs also in the course of the exanthematous fevers, and sometimes it is developed as a sequel of typhoid and typhus fever.

Invasion and Symptoms.—When a child is attacked with this disease the invasion is usually pretty abrupt. In some cases it is marked by the sudden development of a febrile movement. The child, if old enough, may complain of a feeling of giddiness. It becomes drowsy, stupid, and may pass into a comatose condition. It may have repeated attacks of convulsions; it may be delirious; and it may complain of severe diffuse pains in the back and limbs. These constitutional symptoms vary considerably, as they do in intensity, in different cases. In some patients all of them will be present and well defined; in others there will only be a little febrile movement; and in still others there will be a febrile movement, accompanied with some pain in the back and limbs. Again, there may be a febrile movement with a moderate amount of drowsiness, or a little delirium.

If the constitutional symptoms occur they last only a few hours, or two or three days, and do not usually last longer, no matter how severe they may be. The next symptom noticed is paralysis of some of the voluntary muscles, which develops within a few hours, and commonly reaches its maximum within a week. There is loss of power, but not of sensation. The muscles involved may be those of a leg or an arm, or of both legs, and, in the worst cases, of both legs and both arms. The loss of power does not always involve the muscles to the same degree. In a paralyzed limb the extensors will be more involved than the flexors, or the reverse. Occasionally, the loss of power will be more limited than this, and there will not be more than one or two muscles in the arm or leg paralyzed, and those only to a slight extent. Or there may be no paralysis whatever.

In this condition the patients remain for a variable length of time, the paralysis increasing for about a week when it reaches its maximum. After this there is a variable period, during which the condition of the patients remains stationary. Their general health is good enough; they have no constitutional symptoms; they have only a loss of power in some of the voluntary muscles. After remaining in a stationary condition for a few weeks most of the patients begin to slowly improve. In some cases this improvement continues until the muscles have entirely regained their power, and by the end of a year or two years these children may be as well as before the attack. More frequently, however, in children, the recovery is not complete; they get better, but not entirely well; power returns to some of the paralyzed muscles but not to all of them. As a result of this we have various deformities, the children being at an age when the muscles and bones should grow from year to year. They are left with permanent paralysis of some of the muscles of the leg or arm, and, as the paralyzed muscles do not grow as well as the healthy ones, there is, as a consequence, a great variety of deformities. This, then, is the more frequent course of the disease in children.

Occurrence of the Disease in Adults.—In adults the course of the disease is somewhat different. The lesions are supposed to be the same as in children, but there are comparatively few autopsies in adults. In children there have been a good many autopsies. In certain adults affected with this disease its invasion is acute; in others it is chronic. For this reason it is customary to speak of the disease as having both an acute and a chronic form; but the term acute applies to the invasion of the disease rather than to the disease itself. When the disease is once established its course is a chronic one.

Acute Invasion.—In the acute cases the invasion is marked by a febrile movement, vomiting, diarrhœa, colicky pains in the abdomen, headache, a certain amount of drowsiness or stupor, and sometimes delirium and pains in the back and limbs. The latter are occasionally of a dull, diffused character; and at times, on the other hand, of a well-defined neuralgic character, running along the course of the nerve or skipping from one part of the body to another. There may be a pain in the leg, then in the arm, and afterward in the thorax; and so these neuralgic pains may continue to occur rapidly in different parts of the body. There are also disturbances of sensation, the patients complaining of a feeling of numbness in the hands and feet, or, of prickling sensations, as if pins and needles were being thrust into the hands and feet. Tactile sensation is not changed in most cases; in some, however, it is slightly changed.

These symptoms last for a few hours or days, and are followed by a loss of power in some of the voluntary muscles. These acute symptoms may be repeated from time to time. Whenever they subside, nothing abnormal remains except a loss of power in the muscles. After the lapse of weeks or months the constitutional symptoms may return, and, when this is the case, they are followed as before by a fresh loss of muscular power.

The constitutional symptoms vary considerably. In some cases all the symptoms I have mentioned will be present; in others there will be none but the febrile movement; in some, again, there will be a febrile movement and disturbances of sensation. And so the cases vary as to the intensity of the constitutional symptoms which mark the invasion of the disease.

Chronic Invasion.—In chronic cases there is no such sudden invasion, no febrile movement, no cerebral disturbances; but the patients usually suffer from indigestion, or some form of dyspepsia. They are apt to complain of a feeling of weariness in the limbs, and may have disturbances of sensation and neuralgic pains. After these symptoms have lasted for some time, loss of power in the voluntary muscles comes on, and then the disease is fairly established.

Symptoms and General Course of the Disease.—Whether the disease begins acutely or chronically, there is partial or complete paralysis of a certain number of the involuntary muscles. This occurs most commonly in some of the muscles of the legs, and may remain confined to these. In other cases it extends upward and involves one or both of the arms in addition to the legs. In others it involves the arms, afterward extending downward to the legs. In still other cases, only one of the arms or legs is involved, the rest of the extremities escaping. Again, one leg or one arm upon the same side of the body may be involved, which constitutes hemiplegia.

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As in children, so in adults, the muscles are involved unequally, either the flexors or extensors being paralyzed to a greater degree. The muscles of the face and those of the bladder and rectum are not, as a rule, implicated in this disease. Sometimes, however, there will be retention of urine for a few days; most commonly not longer than this. There is not, so far as I know, any incontinence of urine, but there is often stubborn constipation. Paralysis of the voluntary muscles is developed with different degrees of rapidity in different cases. In some instances it only takes a few hours for complete paralysis to develop. Complete paralysis of the legs, or arms will occur in a few hours, and afterward there will be an extension of the paralysis either upward or downward, but subsequently its progress is slow. In some instances it requires months or years before the paralysis reaches its height; in others there are exacerbations of the symptoms. After each return of the constitutional symptoms there will be a decided increase in the paralysis, the patient being left in a worse condition, after each repetition of the constitutional symptoms.

In most cases the paralysis increases, either slowly or rapidly, to a certain point, beyond which it does not pass. This may require hours, weeks, or months, or as much as a year. The patients remain in a stationary condition for a certain length of time, generally for several months. Subsequently there is a gradual improvement, the paralysis beginning to slowly diminish.

In cases of paralysis the texture of the paralyzed muscles is changed. They become soft and flabby, there being always some atrophy which occurs quite rapidly. There is loss of faradic and galvanic contractility in these muscles. In a certain number of cases the reflex action is unchanged; in others it is diminished; and in still others it is increased. Tactile sensation remains normal, or slightly changed. It may be diminished, but never very much. Disturbances of sensation, however, exist in the larger number of cases, such as a prickling sensation and neuralgic pains. There may be days or weeks during which there are no such neuralgic pains, and then they may occur. In many cases very severe attacks of neuralgic pains may be followed by an increased loss of power in the muscles, or by a loss of power in a new group of muscles, or in one muscle. There may have been paralysis of the muscles of the legs, and, after the occurrence of neuralgic pains, the paralysis may extend to one of the arms. Subsequently atrophy of this arm will take place. The intellectual condition of these patients remains good, except when suffering from the febrile movement and constitutional symptoms. These patients do not suffer from bed-sores, and in this respect they differ from those suffering from myelitis.

After the disease has lasted for a certain length of time the patients begin to improve, which may, in some cases, end in complete recovery; but it is a long while before this point is reached. It may require two, three, four, or five years before the paralyzed muscles return to their natural condition. The improvement is progressive, but it is in most cases exceedingly slow, so that the whole course of the disease, including its invasion, the period of development, and the period of recovery, will extend over several years. In other cases, as in children, recovery will not be complete. Some of the paralyzed muscles will return to their natural condition, but there will be groups of muscles, or single muscles, left permanently paralyzed or atrophied. Permanent paralysis and atrophy of the muscles in adults is not nearly so serious a matter as in children, for in adults the development of the muscles and bones is already complete. Hence in adults this condition does not lead to the deformities common in the case of children. The patients are tolerably well, in spite of the loss of power and atrophy of the voluntary muscles.

Treatment.—As regards the treatment of this disease we are aided by our knowledge of its ordinary or natural course. Its natural tendency is to end in recovery, after a time, either completely or in part. Our aim should be to render the patients comfortable, and thus hasten their recovery. The severe neuralgic pains can be controlled by opium. They are so severe and excruciating in their nature that nothing else will answer the purpose. The other constitutional symptoms require no special treatment. The derangement of the stomach and constipation of the bowels should receive special attention. The bowels should be kept open, and the digestion improved as much as possible. While the disease is developing there seems to be an advantage in the persistent use of dry cups along the spinal columns. When it has passed the progressive stage there is an advantage in the systematic use of the faradic current, and in the systematic rubbing of the affected muscles. A weak faradic current should be passed over the muscles every day, and in addition they should be rubbed. The employment of electricity and the systematic rubbing of the part are not to be resorted to until the progressive stage of the disease is past. There is nothing further to be said about treatment.

LECTURE XVII.

DISEASES OF THE SPINAL CORD—CONCLUDED.—LOCOMOTOR ATAXIA.

Locomotor Ataxia; Pathology, Etiology, Symptoms of First, Second, and Third Stages; Prognosis, Treatment.

LOCOMOTOR ATAXIA.—*Pathology.—Gentlemen:* We will take up to-day the consideration of locomotor ataxia. In this disease the lesion consists of a new growth of the interstitial connective tissue of the spinal cord, which is accompanied with atrophy and destruction of nerve-fibres. It has always been a question which of these two conditions is primary, and it is still unsettled. The practical importance of this question is not very great, however, for, as a matter of fact, we always find the two conditions together. The lesion begins in the lateral part of the posterior columns of the cord, and usually in the upper lumbar and lower dorsal regions, and extends both upward and downward. After it has existed for a certain length of time, it extends laterally and involves the lateral parts of the posterior columns, and the columns of Goll. From here the lesion extends to the posterior part of the lateral columns of the cord, and then to the posterior gray cornua, and it may extend even to the anterior gray cornua. When the case has lasted for a very considerable length of time, and the lesion is far advanced, we find the whole cord smaller than normal. There is a production of clear serum between the dura mater of the cord and the cord itself, filling the gap produced by the general atrophy of the cord.

Etiology.—As to the causation of the disease we are very ignorant. We know that it occurs more frequently in males than in females, and usually between the ages of twenty and fifty years, but besides this we know very little. Some patients will give a history of great mental exertion; others will have indulged too freely in sexual pleasures; or they will have been exposed to cold; or, again, there will be a history of too great indulgence in tobacco or alcohol. The disease has also been observed to follow a fall, producing an injury of the loins. Then there are cases in which none of these causes exist, so that we are left in the dark as to the real cause of the disease.

Symptoms: First Stage.—In speaking of the symptoms it is customary to divide them into three stages. During what is called the first stage of the disease, the characteristic symptoms are certain disturbances of sensation, the existence of neuralgic pains, loss of vision, and certain affections of the ocular muscles. The neuralgic pains of locomotor ataxia are of a somewhat peculiar character, and they belong to two categories. In the first place there are pains which follow the course of different nerves. These occur usually in the arms or legs. They come on very suddenly and are exceedingly severe. They may last only for a few minutes, or they may continue for hours and even days. But these prolonged attacks of pain are made up of a succession of short pains shooting down along the course of the nerve. Hardly any pains which human beings suffer are more severe than those that belong to locomotor ataxia. In other cases, instead of the pains following the course of the nerves, they are located in circumscribed parts of the body. It may be in a part of the arm, or leg, or thorax. The affected area is of a few inches in extent, and the nature of the pain is severe and excruciating. These pains are paroxysmal, and are apt to be of longer duration than those confined to the course of the nerves. The pains, either lanceolating or circumscribed, are situated most frequently in the extremities. They may be located in the back, sometimes in the walls of the thorax, and least frequently in the head and face. Occasionally they will be referred to one or other of the viscera, especially to the stomach, intestine, and bladder. When patients suffer from attacks of neuralgic pains in the stomach there are very apt to be, at the same time, disturbances of its functions, these pains being characterized by vomiting. So with pains in the intestine, its function is apt to be interfered with, and patients accordingly suffer from temporary diarrhœa. When they occur in the bladder there may or may not be a change in its capacity for retaining urine; there may or may not be frequent and painful micturition, somewhat like that belonging to cystitis. At the commencement of the disease, the attacks of pain, no matter where situated, do not come on very often, nor do they last for a great length of time. A patient may have an attack of pain, lasting for a few minutes, after which time he will be free of it for one, two, or three months, when he will have another attack. But as the disease advances, the attacks become more frequent and of greater duration.

The affections of the eye are of two kinds: changes referable to the muscles of the eye and changes referable to the optic nerve. In the optic nerve there is developed, in a certain number of cases, lesions of the same character as in the spinal cord—that is, there is a growth of new connective tissue accompanied by atrophy of the nerve-fibres. As a result of this the patients gradually lose their eyesight. These changes may affect one or both eyes in the same patient. The affection of the muscles of the eye consists in loss of power in some of them, in consequence of which the patients have either double vision or strabismus. This paralysis of the recti muscles may be temporary or permanent. Double vision or strabismus may last for a few days or a few weeks, after which time the muscles move as well as ever. Then, after a longer or shorter period, the same thing will happen again, and, in a certain number of cases, this condition will become permanent. In some cases there are changes in the muscles of the iris, resulting in either permanent or temporary dilatation of the pupil of one or both eyes. In still other cases the pupil will be unnaturally contracted, and this will obtain in one or both eyes, so that it does not respond to the light as it should. There is a loss of reflex mobility of the eyes. Then, again, there are other cases in which the pupil remains of natural size, but does not respond to light in the normal way.

The disturbances of sensation consist of certain peculiar feelings, referable usually to the hands and feet—earliest in the feet. Patients will sometimes tell you that they have a feeling of numbness in the feet, as if they were asleep. Others, again, complain of a prickling sensation in the feet. Some patients will tell you that when they put their feet upon the ground they experience a sensation as if there was some soft substance between the feet and the ground, and on this account they do not feel the foot upon the ground as they should. In other patients there will be a feeling as if a girdle was placed about their waist, as is found in cases of myelitis. Again, in some patients there will be patches of skin in different parts of the body which are anæsthetic or hyperæsthetic. Perhaps there will be a place upon one of the thighs where the skin will be either hyperæsthetic or anæsthetic, or there may be several such patches upon the body at the same time. Other patients complain of an unnatural feeling of fatigue; their muscles becoming much more quickly tired than normal. Some patients have a well-marked change in the sexual function. They do not have erections and cannot copulate. They may sometimes suffer from too frequent seminal emissions. These comprise pretty much all of the disturbances of sensation with which we meet.

The first stage of the disease lasts sometimes for months, and occasionally for a great many years; it will be five, ten, fifteen, or twenty years before any other symptoms are developed, but the patients continue to suffer from the symptoms enumerated. During the first stage the nutrition, the mental faculties, and the general condition of the patient will be good. Mental anxiety and severe pain may have some effect upon the general health, but most commonly not a great deal.

Second Stage.—When we come to the second stage of the disease we find that the symptoms which belong to the first continue, but that in addition to these there are others. The most important of these are disturbances of the co ordinate power, by which the action of the voluntary muscles is regulated. These muscles can no longer be moved together in the natural way. Those which are first affected most commonly belong to the leg, and, as a result of this, there is a change in the walk of the patient. He will be able to walk, but no longer in the same regular and easy way that a healthy person does. His legs are moved in an irregular and jerky manner. He will throw out his feet forcibly and bring them down heel first upon the ground. The result is that these patients have a characteristic walk. For a considerable time it will be found that the muscles retain their full power. If the patient sits down and you try to bend his legs against his will you will find no loss of power. The patient has simply lost the faculty of regulating the action of his muscles. After a time this difficulty in coördination of the muscles gradually increases and it may finally become so great that the patient will be unable to walk at all, not because the muscles of his legs are weak, for they may still retain a very considerable amount of strength, but because of his total inability to regulate the movements of his feet and legs. He cannot make his leg move backward and forward as he wishes. He becomes paraplegic not from loss of power but from loss of capacity to regulate the movements of the muscles of his legs. Not infrequently the loss of power extends upward and involves the muscles of the arms, so that the patient moves his arms in a similar manner to his legs, in a jerky, irregular way, and in the worst cases he may finally be unable to move his arms at all, just as he was unable to move his legs.

The disturbances of sensation which belong to the first stage continue during the second. The neuralgic pains continue and are of the same character. Their severity in different cases varies. The feeling of numbness and of pricking, the patches of anaesthesia and hyperaesthesia will continue during the second stage; but there is added to these a permanent loss of sensibility in the affected parts of the body, either in one leg or in both legs and arms. Some patients have a loss of tactile sensation in the skin covering the legs, and occasionally in the skin covering the arms. There is also loss of sensation to painful impressions.

The changes in the muscles of the eye may still continue, and those in the optic nerve become more marked, the patients going on to become totally blind from these causes.

The changes in the bladder become of a more definite character. The patients are now very apt indeed to have either retention or incontinence of urine, and this condition remains permanent, so that the patient is obliged to have his water drawn off by a catheter regularly. The loss of sexual power also becomes much more marked during the second stage than it was during the first stage of the disease. Usually there is complete loss of this function. The mental faculties of these patients are, however, apt to continue good even during the second stage of the disease.

There is still another symptom which may be developed during this stage. It consists in a peculiar affection of the joints. The joint which is most frequently affected is that of the knee, and next in frequency is that of the hip; but all the joints may be affected in the same way. The affection consists in a sudden swelling of the joint, but this is not accompanied with either pain, heat, or redness. The swelling is due to the accumulation of serum in the synovial sac. It may last for a few days or weeks, and disappear entirely, or it may return after the lapse of some time. A single joint may be thus affected a number of times. Upon the disappearance of the swelling the movements of the joints are as good as before. In other cases the swelling is permanent, and, after a time, in addition to the serum in the synovial sack, there is a destruction of the articular ends of the bones composing the joint. They gradually undergo necrotic changes, and when this happens the mobility of the joint is impaired. In the worst cases the destruction of the ends of the bones is so great that dislocation takes place. The ends of the bones lose their normal relations to each other, and the joint becomes entirely useless.

To the second stage of the disease also belongs the change in the reflex action of the muscle. It is best seen in the muscles of the leg and thigh. If we cause a patient to cross one leg over the other, so as to leave his leg and knee free, and strike the point below the patula, there is no action of the muscles, as is the case in health. The involuntary contraction of the muscles is absent. If the same thing be done to a person in health, there is a forcible, quick, and involuntary action of the muscles. This loss of reflex action can also be well shown in the muscles of the calf of the leg. This symptom belongs to the second stage of the disease, although sometimes it is found in the first. In other cases it is not developed until the later part of the second stage. The second stage may continue for a long time, even for years.

Third Stage.—If the patients continue to live, they finally pass into the third stage, and this corresponds to the more general invasion of the spinal cord; to the period of the disease when the changes are no longer confined to the posterior columns of the cord, but have extended to the gray matter and lateral columns. We now have actual loss of power in the affected muscles, due to the invasion of the gray matter and the lateral columns. The muscles which have been previously affected now become completely paralyzed. Besides this there is also actual loss of sensation, which may be partial or complete. The paralysis of the rectum and bladder may become complete, and the patients finally pass into a condition of complete paraplegia, identical with the condition of those patients suffering from ordinary transverse myelitis. The course of the whole disease, including all the different stages, is always protracted. The disease does not prove fatal. If the patients die it is commonly from some intercurrent disease, or if they live until the last stages of the disease they die, as patients do, of myelitis, from bed-sores, cystitis, or pneumonia. The process is a long one, but it is apt to be interrupted. There may be periods of months or even years, during which the symptoms either get no worse, or, if it is in the first stage, they may disappear for a time; or this may happen even in the second stage. These remissions are characteristic of the disease; they are, however, most commonly only temporary.

Prognosis.—The natural tendency of this disease is for the patient to gradually get worse and worse as more of the spinal cord becomes involved. The prognosis is always rendered uncertain by the remissions. A patient may be better for so long a time as to pass from under notice, and you may think he is well; but you may hear of him months or years afterward, and learn that the disease has redeveloped, the patient having passed through one of these remissions. It does seem possible for these patients in some instances to get well, but these are exceptions to the rule.

Treatment.—The plans of treatment which have been adopted for the relief of this disease are very various. They vary a great deal from year to year. Ergot has been used considerably, and commonly in the form of the fluid extract in drachm doses; its use may be continued for a considerable length of time. The bromides have been very extensively employed. The bromides of potassium, sodium, and ammonium have all been used either separately or together. Here, again, any drug, to be of service, must be administered for a long time. The nitrate of silver has also been given in pill form, in doses of from one-fifth to one-fourth of a grain, three times a day, after meals. Besides these drugs, which are the ones most frequently used, the actual cautery has been applied along the course of the spinal column a number of times at intervals; and electricity, in the form of the faradic or galvanic currrent, has also been employed. Good results have been reported from all these methods of treatment; but in spite of this we seem to fall back on the general conclusion that cases of locomotor ataxia do not get well under any plan of treatment. We have now finished the diseases of the spinal cord.

LECTURE XVIII.

DISEASES OF THE THROAT.—PHARYNGITIS.—TONSILLITIS.

Acute Catarrhal Pharyngitis; Pathology, Etiology, Symptoms, Prognosis, Treatment.—Acute Tonsillitis; Pathology, Symptoms, Prognosis, Treatment.—Croupous Tonsillitis; Pathology, Etiology, Symptoms, Prognosis, Treatment.

ACUTE CATARRHAL PHARYNGITIS.—*Gentlemen:* To-day we will begin the consideration of diseases of the throat, and the first of these to which we will devote our attention is *acute catarrhal pharyngitis.*

Pathology.—In this disease the lesion consists in an acute catarrhal inflammation of the mucous membranes covering the throat, the posterior wall of the pharynx, the soft palate, part of the hard palate, and frequently the mucous membrane which covers the posterior nares.

In different attacks of the disease we have the inflammation more or less general. In some cases it is confined to the posterior wall of the pharynx ; in others it is confined to the soft palate ; again, it may be limited to the posterior wall of the soft palate ; while again it may involve the whole tract of the posterior nares, together with the posterior and anterior surfaces of the soft palate. The membrane becomes swollen and congested, and there is an increase in the production of mucus. The amount of swelling, congestion, and increase of mucus will vary a good deal in different cases.

Etiology.—The causes of this disease seem to be purely climatic. There are places where the disease is very common. New York, for example, is a very favorable site for the disease ; many cases of acute pharyngitis occurring throughout the winter, but in the summer there is very little of it. Sometimes, however, the disease seems to be due rather to disturbances of the stomach than to exposure to the weather.

Symptoms.—In its severer forms the disease begins with a well-marked chill ; there may be a single rigor, or it may be repeated during a number of hours. The rigors are followed very rapidly by a febrile movement ; and this, especially in children, may be very well defined, the thermometer indicating a temperature of 103°, 104°, or 105°. There is also very frequently a feeling of pain and discomfort throughout the body, and aching of the back and limbs. These constitutional symptoms are apt to precede the local symptoms.

The local symptoms proper are swelling of the throat, increased production of mucus, with a certain amount of pain, especially when any of the muscles of the throat are moved, as, for instance, in the act of swallowing. This local trouble and the appearances due to the congestion commonly follow the constitutional symptoms, sometimes only by a few hours, or it may be several hours before you can tell that the patient is to have sore throat. In other cases the local symptoms occur without the constitutional symptoms. There may be no rigor, no febrile movement; the patient will feel tolerably well, and simply complain of the throat being sore, and when you look at it you will find the characteristic appearance of the disease. There are exceptional cases in which the appearances of the throat are somewhat different. The mucus may be so great that we may be in doubt as to whether it is croupous inflammation or diphtheria. These cases occur, so far as I know, only in children. The constitutional symptoms, rigors, febrile movement, and prostration are usually well defined. The patients complain of a feeling of soreness in the throat, and upon examination a few hours afterward it is found swollen, congested, and covered with something which looks like false membrane ; a white, tenacious material, which is commonly found upon the posterior wall of the pharynx and soft palate. Although it appears something like a false membrane, it does not look exactly like this. Observing this for the first time you will probably mistake it for false membrane, but will not be likely to err in this respect a second time.

After the disease has existed for a day or so this material begins to disappear, and as it does so its appearance is still more characteristic, being that of long strings of coagulated mucus.

The ordinary duration of the disease is from four days to a week, at the end of which time both the local and constitutional symptoms should disappear. The constitutional symptoms, as a rule, disappear first, the local symptoms later.

Prognosis.—The prognosis of the disease is altogether good. In a certain number of cases the patients are not confined to the bed, but there are a considerable number who, while the disease lasts, appear quite sick, and may be confined to the bed for several days.

Treatment.—The treatment is partly general, partly local. It is well enough, in many cases, to begin the treatment with a mild purgative, and for this purpose there is, perhaps, no better one than calomel, in doses of from five to twelve grains, according to the age of the patient; combine with it a certain amount of opium, so that the patient will be kept quiet at the same time. The bowels are to be kept open. After having done this, perhaps the most efficient drug for the relief of the disease is aconite, given in doses of from half a drop to two drops hourly or every two hours, if you are able to watch the patient. Aconite is a drug of considerable power, and it does not do to give it every hour unless you are able to watch the patient, as he may take too much. A very good way of taking the drug is to put one or two drops of the tincture into a dessertspoonful of liquor ammonii acetatis, repeating this every hour or every two hours. These are the two drugs most used. The local treatment consists of the use of either vapor spray or gargle. Generally, at the beginning of the disease, you will accomplish more by the use of the vapor. You may use the steam of boiling water, adding to the water a certain amount of common vinegar, which is impure acetic acid, or you may use cubebs or a little opium. There is a great variety of drugs of a soothing character which may be used for local medication. During this stage nothing but soothing remedies are applied. Caustics should not be used. After the disease has lasted a few days, and the inflammation has begun to subside, you may begin the use of astringents, which may be applied by means of the spray or gargle. There are a variety of these astringents, all of which answer very well, as alum, borax, sulphate of zinc, and tannic acid. These astringent substances act well in the later stages of the disease, but in the earlier stages they do not act well. A majority of the cases terminate in complete recovery; but a certain number of them do not recover completely, the patients going on to have chronic pharyngitis, which may last for a very considerable length of time.

ACUTE TONSILLITIS.—There is another disease of the throat which resembles acute pharyngitis very much, and this is *acute tonsillitis*.

Pathology.—In acute tonsillitis the part of the tonsils which is involved is the mucous membrane which covers them. The tonsils, as you know, are lymphatic glands, having a peculiar structure. The gland, instead of having the shape of an ordinary lymphatic gland, has little depressions over its surface which are called crypts, and which are lined with mucous membrane. In acute simple tonsillitis it is the mucous membrane covering the tonsils which is involved; the gland itself is not involved, or, at least, only to a slight extent; so, although we call this tonsillitis, we do not mean that the tonsil itself is inflamed, but that the mucous membrane covering the tonsil is inflamed. In this disease one or both tonsils are involved, and with it there may be acute pharyngitis. When you look into the pharynx in acute tonsillitis you find one or both tonsils moderately swollen and congested, and very often you will see a number of white spots varying in size from that of a pin's head to that of a pea. These white spots correspond to the crypts upon the surface of the tonsils. If you detach one of these little white spots and examine it, you will find it to be made up of a collection of coagulated mucus, detached epithelial cells, and a few bacteria. Sometimes these spots reach a considerable size, filling the crypts, and extending over the surface of the tonsil to a considerable extent, looking very much like false membrane.

Symptoms.—The symptoms of acute tonsillitis are identical with those of acute pharyngitis. There are the same constitutional symptoms, followed by the same local appearances. Its duration is about the same as that of acute pharyngitis, the disease running its course in from four days to a week.

Prognosis.—The prognosis is as good as in acute pharyngitis, the patients, as a rule, recovering entirely. As is the case with acute pharyngitis, there is sometimes a disposition for the disease to become chronic. There are some persons who, when suffering from acute tonsillitis, curiously enough, have no pain or soreness of the throat. This not unfrequently happens in children. You will be called to see a child who was the day previous seized with rigors, and you find it quite ill, looking pale, and considerably prostrated. The child will not complain of pain, and the mother will have no idea that it is suffering from a sore throat, but will be apt to think the child is suffering from some severe disease, when it is nothing more than common tonsillitis.

Treatment.—The treatment of acute tonsillitis is just the same as that of acute pharyngitis: the same drugs internally and the same local applications.

CROUPOUS TONSILLITIS.—*Pathology.*—Besides acute inflammation of the mucous membrane covering the tonsils, we meet very frequently with what we may call *croupous tonsillitis.* In these cases we find, as before, either one or both tonsils involved. The tonsils are usually swollen to a greater extent than in simple tonsillitis. Their surface is covered with white patches of false membrane. These white spots are sometimes small and sometimes large enough to extend to the adjacent parts of the pharynx. If you strip up a piece of this white substance and examine it, you will find it composed, not of mucus, but of coagulated fibrine, and tangled in its meshes are a certain number of pus-cells and detached epithelial cells.

Etiology.—As to the causation of this condition we do not know very much. There seems to be in some districts certain epidemic influences which cause the disease to prevail more frequently at one time than at another. I saw an example of this disease in a medical man who had been acting as an assistant in an ophthalmic hospital, and who told me that in the hospital, during the summer, there had been a number of cases of so-called diphtheritic conjunctivitis, and he supposed that the tonsillitis from which he suffered was due to the local causes which had produced this condition in the eye. There are some epidemics, as scarlet fever, in which the children will suffer from croupous tonsillitis. Most frequently the tonsillitis in scarlet fever is simple. Again, there will be certain years and certain seasons of the year during which you will see a number of these cases, and then no more for a considerable length of time.

Symptoms.—The symptoms are much the same as in simple tonsillitis, but are more severe and more protracted. The constitutional symptoms are always present and usually well defined. There are rigors, and a febrile movement which is tolerably intense, lasting for several days. A few hours after the development of the constitutional symptoms, the throat lesions will be well defined. One or both of the tonsils will be considerably swollen and covered with one or more white patches of some size. There is also, most commonly, some swelling of the lymphatic glands of the neck. Such a swelling we do indeed meet with, in some cases of simple tonsillitis, but it is apt to be better defined and is more common in cases of croupous tonsillitis.

The duration of the disease is usually a full week. At the end of this time the constitutional symptoms begin to subside, and this happens before the inflamed part of the throat is clear of false membrane. When it has once begun to disappear it does so very rapidly, and the tonsils are left at first a little swollen, but in a short time they return to their natural condition.

Prognosis.—The prognosis in these cases is good, but it is evident that the patients have been suffering from a disease of some little severity. When a patient has simple tonsillitis, and gets well, he is usually over all the symptoms within one or two days after the disease has run its course ; but in croupous tonsillitis this does not commonly happen. The patient is left prostrated to a considerable extent, and is unable to engage in any active mental or physical occupation. It is sometimes several weeks before his general health is fully restored. The disease is very apt, indeed, to be confounded with that one known as diphtheria, and has been mistaken for it. The differential diagnosis between these conditions is sometimes impossible. There is a considerable number of patients in whose cases you will have to wait for a day or so before they develop symptoms from which you can make up your mind as to which disease they are suffering from. This form of disease is, therefore, most commonly spoken of under the name of diphtheritic sore throat, instead of croupous tonsillitis ; but I think it is better to call it by the latter name, on account of its having no real relationship to diphtheria. It is simply a local inflammation of the tonsils, which runs a definite course and terminates in the recovery of the patient.

Treatment.—The treatment is the same as that of simple tonsillitis, or that of acute catarrhal pharyngitis, both as to the general and local measures. Patients do not need stimulation or any other treatment than is employed in simple tonsillitis.

LECTURE XIX.

DISEASES OF THE THROAT—CONCLUDED.—TONSILLITIS.—LARYNGITIS.

Gangrenous Tonsillitis; Pathology, Occurrence, Symptoms, Prognosis, Treatment.—Quinsy Sore Throat; Pathology, Etiology, Symptoms, Prognosis, Treatment.—Laryngismus Stridulus; First and Second Varieties, Symptoms, Etiology, Prognosis, Treatment.—Acute Catarrhal Laryngitis; Pathology, Etiology, Symptoms, Treatment.

GANGRENOUS TONSILLITIS.—*Pathology and Occurrence.*—*Gentlemen:* We now come to still another form of tonsillitis, namely, that which is called *gangrenous tonsillitis.* In this form of tonsillitis the inflammatory process involves not merely the mucous membrane covering the tonsils, but also the tissue of the tonsils to a greater or less extent, and it may extend to the parts of the pharynx adjacent to the tonsils. The disease is rarely primary. It occurs most frequently as a complication of scarlet fever; least frequently as a complication of measles; occasionally as a complication of typhoid fever. At times it does occur as an idiopathic disease, and when this happens it is most commonly in the form of epidemics.

If we look into the throat of these patients after the disease is fairly developed we find one or both tonsils considerably swollen, as well as the mucous membrane of the pharynx, and we see, also, black patches on the tonsils. These black patches represent parts of dead tissue, which, if the patient lives long enough, become separated or slough away, leaving cavities sometimes of considerable size.

Symptoms.—The general constitutional symptoms which accompany this condition are very well defined. There is a pronounced febrile movement at the commencement of the disease, but the most marked symptom is the extreme depression and prostration of the patient. The heart's action becomes rapid and feeblė, delirium develops, and after a little time the temperature, which was elevated, sinks to the normal, or perhaps below the normal. The skin comes cold and pale, and the patient dies in a condition of colla as in peritonitis.

Prognosis.—The prognosis in this form of tonsillitis is very The larger number of patients die after a few days.

Treatment.—The treatment is altogether unsatisfactory. The indications seem to be to stop the gangrenous process going on in the throat. For this, applications of carbolic acid, salicylic acid, bromine, nitrate of silver, and strong mineral acid have been employed, but the results of all treatments are entirely unsatisfactory.

QUINSY SORE THROAT.—There is still one form of suppurative tonsillitis, or, as it is very often called, quinsy sore throat, to which I wish to direct your attention.

Pathology.—In this affection the inflammatory process involves not the mucous membrane of the tonsils, but the tissue of the tonsils. There may, indeed, sometimes be first an inflammation of the mucous membrane of the tonsils for a few days, which will be succeeded by an inflammation of the tissue of the tonsils proper; but more frequently the disease begins in the substance of the tonsils and goes on to the production of pus. When this happens an abscess is formed in the tissue of the swollen tonsils, and this may be of considerable size, involving either one or both tonsils.

Etiology.—This form of inflammation seems to be due to exposure to cold, but there is a marked predisposition to it in certain individuals.

Symptoms.—Sometimes there are both constitutional and local symptoms. The disease begins with rigors, followed by febrile movement of considerable intensity, lasting, as a rule, for several days. There is, at the same time, a certain amount of prostration, but the patients are not always sick enough to remain in bed. The appetite is lost and there may be disturbances of the stomach, such as vomiting.

The local symptoms consist in the swelling of one or both tonsils. They continue to swell, until, finally, it is impossible to get a view of the posterior wall of the pharynx. Along with this there is most commonly a swelling of the mucous membrane of the pharynx, and very often of that of the mouth. There may be also an increase in the function of the salivary glands, so that mucus is produced in larger quantities, and this will assist in plugging the throat, which is already occluded to a certain extent by the swelling of the tonsils. In most cases there is some swelling upon the outside of the neck, which is partly due to the swollen tonsils. And, in addition to this, the lymphatic glands upon the same side may be swollen, or the whole of one side of the neck may be very much enlarged.

The inflammatory process, as a rule, lasts for a week, by the end of which time the abscess has usually reached a point near enough to the surface of the tonsils to break. The pus escapes into the pharynx and is coughed out by the patient. As soon as the abscess breaks the patient begins to feel better, the tonsil diminishes in size, the extreme suffering decreases, and convalescence is very rapid. Not infrequently, however, when both tonsils are inflamed, the disease runs its course in one tonsil and then begins in the other, the duration being prolonged to ten days or two weeks before the commencement of convalescence.

The discomfort and pain caused by this form of tonsillitis are very great indeed. The patients suffer considerably while the inflammatory process is in progress. They are almost unable to swallow, and do so with great pain. As a rule, the breathing is not obstructed in spite of the extreme swelling of one or both tonsils. Rarely, however, the swelling extends upward, and patients suffer from œdema of the glottis. Sometimes the uvula becomes very much swollen, in which case there will be difficulty in breathing.

Prognosis.—The prognosis is, in most cases, pretty good. The patients suffer considerably for a week or two, at the end of which time they begin to get better. If œdema of the glottis occurs as a complication the danger of death is increased, for a certain number of cases thus affected die. The abscess most frequently breaks into the pharynx, but occasionally it may open beneath the skin of the neck, when it will require opening externally.

Treatment.—At the beginning of the disease you should give, as with simple tonsillitis, a purge; following this, for a day or so, you may give aconite, as in simple tonsillitis, but as soon as the inflammation has resulted in the formation of pus it should be administered no longer. The patients suffer so much that it is often necessary to give them opium, especially at night, in order to keep them moderately comfortable. You have usually to give some wash for the purpose of clearing the throat of mucus. Perhaps as good as any is simply a weak solution of alum or borax. The mouth should be wiped out with a wet towel or handkerchief, and the wash used as a gargle; but commonly these patients cannot gargle efficiently, on account of the swelling of the throat. Sometimes the swelling is so great and the pain so severe that you should not wait for the opening of the abscess, but open it by plunging a bistoury into the swollen tonsil. Very often you can see a point of yellow color where the abscess is about to open. This is the place into which the bistoury should be plunged. You should never attempt to open these abscesses until after the lapse of several days, when they are fairly formed. If you cut into an abscess before it is fairly formed you do harm instead of good, for the patient's suffering is increased thereby. It is very seldom necessary to open these abscesses before the fifth day, and it is still better practice not to open them at all if the patients are not suffering much, but this must be determined by the swelling, and the discomfort of the patient. If the uvula becomes very much swollen, as it occasionally does, you should at once scarify it with a bistoury. You can do this either by making a number of cuts into the surface of the swollen uvula, or if the patient looks as if he were going to choke pretty rapidly, you can plunge a bistoury directly into it and slit it down.

INFLAMMATORY CONDITIONS OF THE LARYNX.

LARYNGISMUS STRIDULUS.—We come now to speak of some of the inflammatory conditions of the larynx, and we will first consider what is rather a nervous affection, known usually under the name of *laryngismus stridulus.* It consists of spasm of the larynx, accompanied with stridulous breathing.

First Variety.—Of this affection there are two varieties. In the first variety the affection consists in a sudden spasmodic closure of the glottis. As a result of this the patients suddenly cease to breathe; they do not breathe with difficulty, but stop breathing altogether. When the spasmodic condition of the muscles which have closed the glottis ceases, and the glottis is allowed to open, the patients take a long, deep inspiration, which is accompanied with a peculiar sound. The glottis is not completely open, and as the air is drawn in it produces a peculiar stridulous sound. The affection occurs most commonly in children who are poor, and badly nourished, and especially in rickety children.

There are no constitutional symptoms whatever. A badly nourished child, apparently suffering from no disease except general want of nutrition, will have these attacks suddenly coming on from time to time. The glottis will close; the breathing will suddenly stop; the child will become pale in the face, or a little livid. It will remain in this condition for a few seconds, then take a few stridulous breaths, after which it will return to the same condition as before the attack. These attacks may be repeated very frequently. A child may have a number of them during every twenty-four hours, and may continue to suffer in this way for several weeks, the attacks coming on more or less irregularly. Frequently the attacks may be excited at will. A sudden fright or a sudden shock, as from throwing cold water into the face, or blowing into it, may bring on an attack.

Occasionally, however, closure of the glottis continues and the child dies. These attacks are always alarming. The pulse is barely perceptible, and, although the attack lasts only a few seconds, these seem to be very long seconds, and besides, we know that any particular attack may prove fatal.

Treatment.—The treatment is especially directed to the prevention of these attacks, and for this purpose the one indication is to improve the general nutrition of the patient. You do not give them antispasmodic remedies, so called, to prevent the re-occurrence of these attacks, but you have these children take iron, quinine, or the mineral acids, and, in addition, cause them to be sponged off with cold water every morning. At the time of the attack you usually do nothing, for it lasts only a few seconds, and then passes away. If the attack lasts longer, and it seems as if the child was not going to breathe, of course, the indication is to perform tracheotomy; but, unfortunately, there is, as a rule, no time to perform this operation, for before it can be accomplished the child is either dead or has recovered. Lately Dr. Polk, of this city, adopted an efficacious plan of immediate relief for this condition. He was exhibiting the child to his class, when it was suddenly attacked with spasm of the glottis. The child ceased breathing, and was apparently dead. Dr. Polk, after waiting a few seconds, took an ordinary silver catheter and passed it down into the larynx. There was a deep inspiration, and the child began to breathe. This suggestion is a very valuable one, and it should be remembered that under such circumstances the indications are to pass a small catheter through the larynx into the trachea. As soon as the breathing has commenced the catheter may be withdrawn, for the breathing will go on, the attack being spasmodic.

Second Variety.—The second variety of laryngismus stridulus, although called by the same name, and consisting of a spasmodic affection which closes the glottis, is yet very different from the laryngismus stridulus of which we have just been speaking. In this variety there is a partial closure of the glottis. This is not accompanied by the cessation of the respiratory act, but the breathing is difficult.

This condition does not occur any more frequently in poor and ill-nourished children than in healthy ones. On the contrary, some of the stoutest and best-nourished children are particularly liable to this affection. The attacks do not come on as frequently as do those of the first variety of laryngismus stridulus. The attacks will come on and last for hours, and then disappear and not return for several months, or for a year or so. The attacks are not frequently repeated.

Etiology.—These attacks seem to be due, in some cases, to exposure to cold, in others to indigestion. They are most apt to occur in the night, especially between the hours of twelve and two o'clock. The child may have gone to bed apparently in good health, and will wake up in the middle of the night almost unable to breathe. It will sit up in bed, gasp for breath, move the whole thorax violently in efforts at breathing, and the sound of the breathing will be of a stridulous character; a peculiar hard breathing, which, when you have once heard, you will always be able to recognize afterward. This condition lasts sometimes for half an hour, occasionally for several hours, and then subsides, the child breathing naturally, and by the next day is as well as ever. In some cases the same thing will occur on the following night or it may never occur again.

There are no constitutional symptoms whatever, no febrile movements, no prostration; there is nothing except the local condition of partial closure of the glottis and the difficulty of breathing due to this. As soon as the spasmodic condition subsides, the child continues to breathe as well as before.

Prognosis.—The prognosis of this condition is good, and the patients, as a rule, get well. But you should remember that these attacks are very alarming, and the children very often look as if they were really choking or suffocating; but yet, in spite of these alarming appearances, they do get well.

Treatment.—The indication for treatment is to relieve the spasmodic closure of the glottis as rapidly as possible, and usually the best way of accomplishing this is by the use of emetics. Perhaps the best of these is the yellow sulphate of mercury, given in doses of from three to five grains. In children you can give one such powder, and if they do not vomit within a few minutes a second powder of the same size may be given. This is a very prompt emetic and does not disturb the stomach afterward. When you cannot procure this you may use any other emetic at hand. Ipecac may be given, but it is not so certain in its effects. I have seen children who have taken as much as twenty teaspoonfuls of the wine of ipecac, without having any other effect than producing diarrhœa. The indication is to cause the child to vomit as rapidly as possible. As soon as the child does this, the spasmodic condition of the glottis begins to subside and the patient is relieved. It may happen, however, that it is relieved only for a time—for an hour or so—and then the breathing becomes bad. When the attack is repeated you are to give a second emetic, but it is seldom necessary to do more than this. There is no after-treatment.

ACUTE CATARRHAL LARYNGITIS.—Acute catarrhal laryngitis occurs both in children and adults. The symptoms, however, differ considerably according to the age of the patients. The symptoms of acute laryngitis of adults are different from those in children, but in both the lesion is the same.

Pathology.—The mucous membrane of the larynx becomes the seat of an acute catarrhal inflammation. As a result of this it is congested, swollen, and at first dry. The function of the mucous glands is arrested, and in consequence of this the membrane is dryer than it should be. After the lapse of a varying length of time there is an increased production of mucus, and the larynx becomes coated with it. For some reason the mucous membrane of the larynx is different from most mucous membranes. The rule is, that when a mucous membrane is inflamed it is dryer than normal. After the inflammation has lasted for a few hours, there is an increased production of mucus. But in the inflammation of the mucous membrane of the larynx the function may be arrested, not only for several hours, but for several days. It may be two or three days before there will be an increased production of mucus, and this gives rise to a difference in the symptoms due to the inflammatory process.

The inflammation may be confined to the wall of the larynx, or extend to the trachea, or even down to the bronchi and upward, involving the pharynx and posterior nares.

Etiology.—This disease may sometimes occur as an idiopathic inflammatory process. It also occurs in connection with constitutional syphilis, and with chronic pulmonary phthisis. It occurs as a result of inhalation of hot steam or smoke, or as a complication of measles scarlet fever, small-pox, or typhoid fever. As an idiopathic disease we do not meet with it very frequently. As a complication of syphilis and phthisis, we see it quite often; as a result of the inhalation of hot steam or smoke, we meet with it from time to time.

Symptoms.—The symptoms consist of a certain amount of constitutional disturbance and local manifestations. There is a febrile movement, which, as a rule, is of no great intensity. There is usually no great prostration of these patients, and they do not feel very sick.

The local symptoms consist first of a cough, and this is of a peculiar character. It is a dry, harsh, metallic, stridulus cough, as if produced in a dry larynx, and not in a larynx in which there is any production of mucus. Besides the cough there is pain referred to the region of the larynx, and this may be quite severe. It may be confined to the larynx or extend down into the thorax. The voice is affected, the patients speaking with a harsh, metallic voice, which has the same peculiar quality as the cough; or the patients may lose their voice almost altogether, and only be able to speak in a whisper. Patients also suffer from dyspnœa, which is due directly to the affection of the larynx, the inflammatory process causing a certain amount of obstruction, or narrowing of the passages, so as not to allow the air to pass through, in inspiration or expiration, as it should. The degree of dyspnœa differs very much in different individuals. Occasionally it is only moderate; again it may be very severe, and may even prove fatal. It is not usually continuous, and from time to time it will suddenly become very much worse. The patient will sit up in bed and struggle for his breath; his face will become livid, his skin cold, and the attack will last for a few minutes and then pass off, the patient returning to a condition of ease. Exacerbations of dyspnœa are always very dangerous. It is always possible for the patient to die in them, and that, too, very quickly.

Treatment.—The treatment of this condition consists in attempting to limit the inflammation, to moderate its severity, and to relieve the dyspnœa. The means used to moderate the severity of the inflammation are, in the first place, applications made to the throat. We can apply leeches over the larynx, or cold, by means of compresses, or counter-irritation may be employed. Of drugs we can use those which are likely to increase the production of mucus from the affected mucous membrane; for as soon as the dry stage of the inflammation has passed, and mucus is produced in a considerable amount, the dyspnœa is usually relieved.

The drug which is most efficacious in this respect is possibly tartarized antimony or tartar emetic. You give it in small doses, from one-hundredth to one-fiftieth of a grain, according to the age of the patient. Another drug which has the same property is iodide of potassium, which may be given in five-grain doses; but it is not as good as the antimony. Iodide of potassium will increase the functions of the mucous glands of the larynx in a certain number of persons, but antimony will do it for nearly all.

To relieve the extreme dyspnœa which sometimes exists, you will find considerable benefit from the application of sponges to the nape of the neck; this is frequently more efficacious than you might suppose. If you wring out a sponge from hot water, and apply it to the neck, putting a second sponge in the same place as soon as the first ceases to be hot, and continue the treatment, you will find it to afford all the relief necessary in some cases.

In attending these cases you should always be in readiness for the performance of tracheotomy. Even in a case which is doing pretty well there is always a liability of the patient being attacked with urgent dyspnœa, which nothing will relieve except tracheotomy.

LECTURE XX.

DISEASES OF THE LARYNX—CONCLUDED.—CATARRHAL AND CROUPOUS LARYNGITIS.

Catarrhal Laryngitis, or Croup; Occurrence, Symptoms, Treatment.—Croupous Inflammation of the Larynx; Pathology, Etiology and Occurrence, Symptoms, Prognosis, Treatment.

CATARRHAL LARYNGITIS IN CHILDREN.—*Occurrence.—Gentlemen:* Catarrhal laryngitis in children, or catarrhal croup, as it is sometimes called, is commonly an idiopathic disease. It may occur from exposure to cold or without any known cause. It occurs as a complication of measles, less frequently as a complication of scarlet fever, and in children, as in adults, it may be produced by the inhalation of hot steam or hot smoke, or by the drinking of hot water. Children sometimes have a fancy to drink hot water from the spout of a tea-kettle as it stands upon the stove, and when they do so it is very liable to give rise to the inflammatory affections of the larynx under consideration.

The disease usually occurs between the ages of one and five years, the period of dentition. It is seldom seen in children under one year old, and in most children the attacks do not occur after five years of age; but still we may see it in children of eight, nine, or ten years, and occasionally in those of fourteen or fifteen, and rarely, where there is a predisposition to this affection, it may continue in adult life.

Some children have a predisposition to this affection, and suffer from attacks of it one or more times every winter, so that the parents learn to expect it. Children born of parents who were subject to catarrhal croup in their childhood are very apt indeed to suffer from such attacks. Exposure to cold seems to be a very frequent cause of its appearance in children who have a predisposition to the disease. Such a child, if exposed to cold instead of having an attack of bronchitis, or a cold in the head, or tonsillitis, will be very likely to have catarrhal laryngitis.

The lesions are the same as in catarrhal laryngitis in adults, and, as in adults, the process may extend downward and upward.

Symptoms.—The symptoms of this inflammation are both local and general. The local symptoms consist in stridulous breathing, stridulous cough, and stridulous voice.

In addition to stridulous breathing there may be difficult breathing. The child may suffer from laryngeal dyspnœa, and this may be exaggerated to such an extent as to give rise to the idea that the child is about to suffocate. So you have stridulous breathing without suffocation, and stridulous breathing with a moderate amount of dyspnœa, and stridulous breathing with very great dyspnœa.

The voice may not alone be stridulous, but may be nearly lost, the child being only able to speak in a whisper. As a rule, children do not complain of any pain over the larynx as do adults.

The general symptoms consist in a febrile movement, which is occasionally preceded by a slight rigor. The local symptoms precede the febrile movement, or they may be developed nearly at the same time. The invasion of the disease is, in many of the cases, very sudden. The child goes to bed apparently well, and in the middle of the night, between one and three o'clock, it will wake up with a hard, stridulous cough, stridulous breath, and stridulous voice, and there may or may not be a febrile movement accompanying them. In other cases the child will suffer with an ordinary cold in the head, from one to three days, after which the laryngeal symptoms will appear in the usual way.

There are other cases in which the invasion is more gradual. It will be noticed, in the afternoon of some particular day, that the child is a little hoarse, and that this increases toward evening. The child goes to bed feeling pretty comfortably, but in the night it may get worse, developing the symptoms above mentioned.

Most of the cases last for three nights. During the day the children are commonly better. The second night is apt to be the worst, and the disease is fully established at this time. In the early part of the evening the child will be well enough to be up and to play, but when the night comes on the symptoms become worse. By the third night there is usually an increased production of mucus from the mucous membrane of the larynx. This marks the close of the disease, and the child gets well.

Cases vary considerably as to the degree of febrile movement and the amount of dyspnœa. In some instances neither of these symptoms are well defined, but in others they are very pronounced during the night-time, but not usually during the day-time. It is quite frequent for this disease to be succeeded by bronchitis. There are a few cases in which the disease lasts longer than its regular time. It may continue for a week, or ten days, and yet during all this time it will preserve the same characteristic feature of being worse during the night and better during the day. There are a few cases in which the symptoms are exceedingly severe. Other cases begin like those of catarrhal laryngitis, and in a day or two the character of the inflammation will change and the patient will have croupous laryngitis.

Treatment.—In the treatment of this condition there are several indications. In the first place, the production of mucus by the laryngeal mucous membranes should be promoted, and in this way relieve the child from its stridulous breathing, cough, and voice. The second indication is to quiet the restlessness from which these children suffer, it being most pronounced at night. The third indication is to relieve the severe attacks of dyspnœa in which the patients seem about to suffocate. The febrile movement is not usually severe enough to call for any treatment. To promote the production of mucus we use antimony as before, but instead of using the tartar emetic, as in adults, it will be better to administer antimonial wine. The ordinary dose for children, from two to five years of age, is ten drops every hour, or every two or three hours, according to the character of the symptoms. If you find that ten drops cause the child to vomit, you do not cease to give the medicine, but diminish the dose. If five drops induce vomiting, give from one to three drops, the object being not to cause vomiting but to increase the production of mucus in the larynx.

To relieve the restlessness you are to administer some preparation of opium in small doses. You may give Dover's powder, paregoric, or laudanum, or even syrup of poppies, which contains a very small amount of opium. To relieve the severe attacks of dyspnœa you give an emetic which will cause the child to vomit promptly. For this purpose the yellow sulphate of mercury, of which I have already spoken, in doses of from three to five grains, will be found very efficacious. The syrup of the wine of ipecac in drachm doses, or antimonial wine in drachm doses, will act as an emetic. These are the three indications to be met.

You must remember in the treatment of these cases what the natural course of the disease is. I doubt if there is any disease which most of you will see as much of as this particular one, for it is, perhaps, the most frequent disease of childhood. You should remember that the natural course of the disease is to recover, no matter what you do, and that the object of all your treatment is to relieve the symptoms, that the patient may get well more promptly. You must not get alarmed at the appearance of these children. Parents do become alarmed, and not infrequently this happens to physicians. The consequence is that the patients get treated more energetically than they ought to be. Last winter I saw a child belonging to a family I am in the habit of taking care of. The child was taken sick in the country, and it was kept in bed for a week, and not allowed to have its clothes changed during this time. In the room water was kept boiling, and slack lime was frequently used. By this procedure the child was made moderately ill for a week, instead of being less ill for three days, and it was several weeks before the child entirely recovered. This is not an exception, for the rule is that these children are treated too much, in view of the fact that the disease usually terminates in recovery at the end of three days

In those cases in which the disease is protracted beyond its average duration, you are usually called upon to do something more than in the ordinary cases. These children, as a rule, are not very sick; but still it is an annoyance to the parents if the disease is prolonged. Perhaps as good a way to break it up as any is to give calomel in one-grain doses until the bowels begin to move; most commonly it will not require more than five or six hours to accomplish this. As soon as the bowels have been moved you stop its use, and wait for twelve or twenty-four hours until its effects begin to pass off. Then you should begin with small doses of quinine, from one-fourth of a grain to a grain, according to the age of the child, three times a day. By this plan of treatment these cases can be brought to a successful close. The most severe cases of it can be treated as cases of croupous laryngitis, but these are fortunately very rare.

Croupous Inflammation of the Larynx.—We now come to speak of croupous inflammation of the larynx, or membranous croup, as it is commonly called when it occurs in children.

Pathology.—The lesion consists in a croupous inflammation of the mucous membrane of the larynx. The mucous membrane becomes congested, swollen, and covered with a layer of false membrane, which consists of coagulated fibrine, of pus-cells, of altered epithelial cells, and very often of bacteria. The inflammatory process is in some cases confined to the larynx; very frequently, however, it extends downward and involves the trachea, and may extend even farther down and involve the bronchi, or even the lung-tissue proper, and the child will suffer from pneumonia, or from an inflammation of the same character affecting the air-cells. In other cases the inflammatory process may extend upward, involving the pharynx and part of the tonsils, which are covered in the same way with a false membrane, having the same structure and the same appearance.

Etiology and Occurrence.—The disease is rare in adults, but when it occurs in them it is, as a rule, from traumatic causes, as the inhalation of hot steam, or hot air. Firemen are sometimes attacked in this way when they go too close to hot smoke in burning buildings. Boiler explosions and the inhalation of hot steam from any source may give rise to the disease.

In children it occurs as an idiopathic disease, as a complication of measles and scarlet fever, and as in adults from traumatic causes. Of the idiopathic cases the larger number occur during the second year of childhood, and nearly all the cases occur in children between the ages of two and five years. Most of the cases occur in children two years old. It occurs in healthy children as well as in poor and illy nourished ones, but like most every disease it is more frequent among the poor and those who live in tenement houses. It is a disease of cold and temperate climates, and is most prevalent during the cold and temperate seasons of the year. Very few cases occur in summer. In New York there are always a certain number of cases every year, but whenever the disease known as diphtheria is prevailing it is always difficult to know how many of the cases are those of simple croupous laryngitis and how many are cases of diphtheria.

We experience this same difficulty in regard to other diseases. For example, when cerebro-spinal meningitis is prevailing it is often difficult to say whether the case under observation is one of cerebro-spinal meningitis or simply a local affection. The same is true of diphtheria. When it is prevailing it is often difficult to say whether the patient is suffering from croupous laryngitis or from diphtheria, with the lesion confined to the larynx.

Symptoms.—The symptoms of croupous laryngitis begin in much the same way as those of catarrhal laryngitis, but both the local and general symptoms are more pronounced, as a rule, from the outset. There is a febrile movement usually at the very beginning of the disease. The attack coming on late in the afternoon, or in the evening, does not subside, and on the following day the laryngeal symptoms continue to get worse. There is at first the same stridulous breathing, stridulous cough, and stridulous voice that was present in catarrhal laryngitis. The dyspnœa comes on very slowly and continues to increase. The febrile movement continues, and the whole appearance of the child is that of one being seriously ill. By the end of twenty-four hours the inflammatory products begin to show themselves, but are not at this time present in sufficient quantity to produce a false membrane. If the inflammation extend to the pharynx you may be able to see the products of inflammation, but if it is confined to the larynx you will be unable to do so. The dyspnœa constantly increases, and it is sometimes worse than at other times during the twenty-four hours. After the dyspnœa has lasted for a certain length of time, the symptoms of carbonic acid poisoning begin to show themselves. There is generally a venous congestion and livid color of the skin over the whole body. In other cases, instead of this, the skin is unnaturally pale; the pulse will become more and more feeble, and if the child does not die simply from dyspnœa, it may die from an attack of convulsions, or becoming comatose, will die in this condition. The ordinary duration of the disease in fatal cases is from four to six days. These patients either suffocate or die in an attack of convulsions or coma.

There are a few cases which run their course within twenty-four hours. In these the heart's action comes to be very rapid and feeble. The obstruction to the breathing increases rapidly and the child dies within a short time. It is a peculiar feature of the dyspnœa of laryngitis, that it is not always obvious or apparent. In most cases of dyspnœa, by looking at the patient, you see that there is a mechanical obstruction to the ingress and egress of air from the lungs. This difficulty to the passage of air will be met by endeavors on the part of the patient to fight against it. The child, in breathing, will make great efforts to overcome the obstruction. It will move the whole thorax as vigorously as it can, and under such circumstances the dyspnœa is apparent. Occasionally, however, you will see cases in which the effort referred to is not apparent. The child breathes badly, but makes no effort to breathe any better. The walls of the chest will not move any more than those of a child in good health when breathing quietly, and yet, all the time it is being more and more suffocated. It sometimes happens in these cases that the parents, and even the medical attendants do not appreciate the danger. The child seeming to breathe easily, it is supposed that it does breathe easily, and under such circumstances, not infrequently, the child suffocates ; dying in the same way as those children who fight against the difficulty in breathing.

Prognosis.—The prognosis of croupous laryngitis is bad, the larger number of patients dying. The prognosis is, however, not hopeless, for there is always a small number who recover. Generally speaking, the older the child the better its chance to recover. Children, five or more years of age, are more likely to recover than those of two years of age.

Treatment.—The treatment of croupous laryngitis consists in an attempt to diminish the inflammatory process, to sustain the strength of the patient, and when the false membrane is fairly formed and acts as an obstruction to breathing to any considerable extent, to open the trachea and allow air to enter and to escape from the lungs.

Of the means which may be employed in the endeavor to limit the local inflammation, there are, in the first place, local measures. If the child is strong and robust, leeches may be applied to the neck, in region of larynx. Some years ago it was the habit to employ general bloodletting, but now this is hardly ever done. Hot or cold applications may be used. Either ice, or cloths, or sponges, wrung out of hot water may be applied to the neck. It is customary to allow these children to breathe vapor of some kind ; either that of boiling water, or that which comes from slacking lime, or the water used may be medicated in some way. When this is done it is usually best to fill the room, or if you cannot do this, make a tent over the child and fill this with vapor. The idea of causing the child to inhale vapor, is, that the false membrane will more easily and quickly become detached from the mucous membrane beneath, if the child breathes a moist atmosphere, than would be the case if the child breathed the ordinary atmosphere.

As regards drugs, they are not of much service. Formerly it was customary to give calomel in considerable doses, up to the point of salivation. There does not seem to be any indication to give it up to such a point, but I think, perhaps, there was some reason for the old practice. I think benefit may be derived in giving it in grain or half-grain doses, every hour or two hours, until the bowels are moved, after which cease its administration. The drug seems to have some effect upon the progress of the inflammation. The extreme restlessness from which these children suffer may require the use of opium in small doses. The insufficient æration of blood may be compensated for by allowing the child to breathe oxygen gas instead of ordinary air. After the disease has progressed so far that there is a considerable production of false membrane, if the child breathes with difficulty, and it is evident that the blood is becoming imperfectly ærated, then the question of tracheotomy comes up, and I think in these cases it should always be answered in the affirmative. If the children are suffering, not from general constitutional disease, but from local inflammation, and the difficulty in breathing is due to the mechanical effects of the local inflammatory process, and, it is thought that by the performance of tracheotomy the patient may be enabled to live until the local inflammation runs its course, the operation should be performed at once, for then the children stand a good chance of recovery. Of the children who die after the performance of tracheotomy, the larger number of them die either from the extension of the inflammatory products to the bronchi (croupous bronchitis), or from complicating pneumonia (croupous pneumonia). The liability to croupous bronchitis and croupous pneumonia exist, whether tracheotomy is performed or not. These constitute, then, the remedial measures which we can use to fight against the disease.

LECTURE XXI.

DIPHTHERIA.

Diphtheria: Meaning of the Word, Pathology, Occurrence, Etiology, Communicability, Nature, Symptoms in Malignant Cases.

DIPHTHERIA.—*Gentlemen:* We come now to the consideration of another of the general diseases—a general disease having a characteristic local lesion which is now commonly known under the name of *diphtheria*. This name has been given to the disease since 1820, but the disease itself has apparently prevailed for a much longer time. The noun diphtheria and the adjective diphtheritic are not always used with the same meaning. You will probably be more or less confused by the different ways in which these terms are used in different text-books. The word diphtheria is derived from a Greek word which means a membrane, and for this reason the terms diphtheria and diphtheritic have been applied to a number of conditions in which a new membrane is produced. The form of inflammation I am in the habit of describing as croupous, is very often defined as diphtheritic, simply meaning an inflammation attended with the production of false membrane. Still more than this, the term diphtheritic is sometimes applied to catarrhal inflammations where there is a good deal of mucus formed, as in tonsillitis, where we have patches of mucus of considerable size. This is spoken of sometimes as diphtheritic sore throat, or diphtheritic tonsillitis. Besides this there is the general disease diphtheria, which has a characteristic appearance.

Pathology.—The lesion of diphtheria consists in a croupous inflammation of some of the mucous membranes, especially those covering the tonsils, pharynx, nares, soft palate, larynx, and trachea. In some cases the lesion will be confined to one of these membranes; in other cases they will all be involved. In still other cases the lesion will begin at one particular point, and extend to involve a larger and larger area of mucous membrane. Less frequently the mucous membrane of the mouth and gums becomes inflamed. In the same way the mucous membranes of the œsophagus, stomach, and vagina may all become the seat of a similar lesion, and if the skin is abraded it may be covered with a layer of fibrine and pus.

The croupous inflammation of these membranes has the character of ordinary croupous inflammation. There is produced upon the surface of the inflamed membrane a false membrane, which is composed of coagulated fibrine, pus-cells, epithelial cells, and bacteria. The stroma of the mucous membrane is infiltrated with pus and fibrine to a greater or less degree. The extent of the lesion varies considerably in different examples of the disease, even when it proves fatal. Sometimes there is general congestion and swelling of the mucous membrane, with the formation of a very little false membrane; and the false membrane which we do find is not continuous, but is present in the form of circumscribed patches. A considerable extent of the pharynx, tonsils, larynx, and trachea may be inflamed, but not covered with a continuous layer of false membrane. Here and there small patches only will be found.

In some other cases a considerable area of mucous membrane is inflamed, and it is covered with fibrine, but this may be so scanty as hardly to be seen, the appearance being that of general congestion and swelling of the parts. If the patient dies it will be found that the inflamed membrane is coated with a very thin layer of fibrine, not thick enough to be distinctly seen with the naked eye, but still enough to be appreciated. In other cases the false membrane is produced in considerable quantity and is continuous, and the whole of the tonsils, pharynx, posterior nares, trachea, and larynx may be covered with a continuous layer of false membrane. Then there are some other cases which are characterized by an infiltration of the connective tissue with the products of inflammation, and in these the general swelling of the parts becomes much more marked. There is not only a thick false membrane upon the surface of the mucous membrane, but the tissue itself is infiltrated to a great extent. In other cases gangrene is added to the inflammatory process. This takes place when the inflammation of the submucous tissue is very great on account of the parts losing their vitality. The sloughs formed in this way may be of considerable size. In some cases there is swelling of the cervical and lymphatic glands. Not only these, but the salivary glands may become swollen, and when this takes place they can scarcely be distinguished one from the other. The swelling of these glands is succeeded by suppurative inflammation, so that pus is formed in the neck and may be discharged externally through the skin.

Complications.—There may be ordinary bronchitis and ordinary pneumonia as a complication of the disease, or the bronchitis may partake of the same characters as the primary inflammation of the throat, or it may be a croupous bronchitis. The pneumonia is sometimes croupous and at other times of the lobular variety. The kidneys are very often the seat of parenchymatous inflammation, of an inflammation confined to the epithelial cells which line the tubules of the kidney and the capsules of the Malpighian bodies.

Occurrence.—The disease has prevailed in the form of epidemics for a great many years. Our more exact knowledge dates from the year 1820. Since that time epidemics have been more closely observed. When the disease once occurs as an epidemic, especially in cities, it is apt to remain as an endemic disease. This has been the case in New York. The disease prevailed as an epidemic some years ago, and it has continued as an endemic disease ever since. This fall it has assumed very considerable proportions, and the number of cases has been very large indeed. The disease has been observed in almost every part of the world and in almost all climates; but it is observed much the most frequently in temperate climates. It prevails very frequently in those parts of the country where the land is low and the soil damp. It occurs both on the sea coast and in the interior. There seems to be no very great preference as to the season of the year in which it occurs. The larger number of epidemics have occurred in the spring, but the disease occurs at all seasons of the year. In the chart before you, which represents the mortality in New York from this disease between the years 1871 and 1875, you will observe that the disease shows no particular preference as to season, the cases being distributed through the different months quite regularly.

Etiology.—As regards the causative influence of defective drainage or sewerage, the inhalation of the air of crowded buildings or the air of privies, and so on, our information is not as yet exact. We have no definite knowledge as to the influence of these conditions in producing the disease, but a considerable number of cases have been observed where the drainage was bad, in houses having filthy privies, in badly ventilated houses, and in low, damp districts of country. We find patients with this disease, however, in houses where the drainage is perfect, the air good, and where there seems to be no local condition that might account for its origin.

The disease, in the majority of cases, occurs among young children; but there are some epidemics in which as many grown people as children are attacked, and there have been epidemics in which children have escaped the disease entirely, the only cases being among adults. In New York the disease is most frequent among children, but it is not by any means confined to them. There have been a considerable number of cases among adults, but much the larger number occur in children under fifteen years of age.

Communicability.—It seems tolerably certain that the disease is directly contagious; that it can be transmitted from one person to another. Exactly how this is effected we do not know. It is possible that the contagious material is derived from the local lesion alone; but it is also equally possible that it is derived in some way from the whole body, as in scarlet fever, small-pox, and measles; for it seems to be contagious in about the same way as the three last-mentioned diseases. Fortunately it is not a very contagious, but rather one of the less contagious diseases. A considerable number of persons may be exposed to the poison and escape being affected. A person may be in the same room with a patient suffering from the disease and not contract it. Children are very much more liable to take it under such circumstances than are adults. It also seems tolerably certain that the disease is capable of originating spontaneously, that is, we can find no cause for its existence. There must be a cause, but what it is we do not know. We can guard against most contagious diseases, but against diphtheria we have as yet no safeguard.

Nature.—As regards the nature of the disease there is still great difference of opinion. It it believed by some, and I think correctly, that it belongs to the class of general infectious diseases, like typhus and typhoid fever, or cerebro-spinal meningitis. There is also a belief that the disease is at first simply an inflammation, purely local, and of a peculiar specific character. As a result of this specific local inflammation the whole system becomes poisoned, and the patients go on to suffer from a general disease. There are certain modifications of these two general ideas as to the nature of the disease. Most people believe that it is either a general disease, characterized by a local lesion, or that it is a specific, local inflammation, which leads to a general poisoning of the whole system.

Symptoms in Malignant Cases.—When we come to the symptoms of the disease we find considerable variety in the cases in different epidemics, and even in the same epidemic. In the first place there are some cases which may fairly be called malignant, on account of the severity of the constitutional symptoms, and the rapidity with which death is produced. Of these malignant cases there are two kinds, which run a very different course. They are equally malignant, but the condition of the patients is very different.

In the first variety of the malignant cases the invasion of the disease is croupy, the patients suddenly becoming victims of a marked febrile movement, which may be preceded by rigors, and may be accompanied by one or more convulsions, especially when the disease occurs in children. The febrile movement continues, and the temperature runs up to 104° or 105°. The pulse is at first full and rapid. The local lesion is developed very rapidly, and in the course of a few hours one of the tonsils may be swollen, or there is general swelling of the whole pharynx and the tonsils. By the end of twelve or twenty-four hours the false membrane will begin to form, sometimes upon one of the tonsils, or it may begin to form at some particular point in the pharynx. The febrile movement and local inflammation continue, and more false membrane is constantly produced over a larger and larger area, until the whole of the pharynx, soft palate, posterior nares, larynx, and bronchi may be covered with a continuous layer of thick false membrane. In some cases of this form of the disease there is the general swelling of the neck of which I have spoken as being due to the enlargement of the cervical and lymphatic glands. When this is extreme, as it is occasionally, the patient's head will be thrown back as in opisthotonos. Most commonly the urine contains albumen, and becomes diminished in amount; it may be almost suppressed. It may contain a few hyaline and granular casts. The patients develop cerebral symptoms, become delirious, drowsy, comatose, and die in this condition, usually at the end of three or five days. The progress of the disease is nearly continuous, the patient getting worse from hour to hour and from day to day.

In the second set of malignant cases the invasion of the disease is not acute, it is insidious. The child at first simply seems to be a little out of sorts. It loses its appetite and does not wish to play. It does not care for anything except to sit still or lie upon the bed. There is no febrile movement, and the child does not complain much. If you look at the throat you will see nothing abnormal, except, perhaps, there may be a little congestion. The next thing which is observed is that the neck becomes somewhat swollen, which is due either to the swelling of the lymphatic or the salivary glands. Should you now look at the throat you will find a very marked congestion, but still the child will not complain of its throat. It may complain of the swelling of the neck, but will say that there is no pain. The pulse becomes more frequent and feeble, the skin is pale and cool; there begins to be a discharge of dirty, brown fluid, which runs from the mouth and nose, especially when the child is lying down, and when asleep. If you look at the pillow you will find it discolored by this thin fluid. The child may continue in this condition, without any more marked symptoms, for three or four days or a couple of weeks, and during all this time there will be no febrile movement, no false membrane formed. Nothing will be observed but the general depression of the child, the heart's feeble action, and the escape of the dirty, brown fluid. In addition, the urine may contain albumen. At the end of three or four days, or two weeks, the child dies suddenly. You may see it in the condition I have just described, the child not appearing to be very ill, and yet within half an hour it may suddenly die. The child's breathing will become a little rapid, then stop, and it will be dead. Occasionally these children die so suddenly that the people who are taking care of them are not aware of it. The child may be lying in bed by the side of its mother or nurse, and she may wake up in the night, look at the child, and find that it does not move. It feels cold, is rigid, and has evidently been dead for some time. Death occurs in a sudden and unexpected way. Although there is no false membrane, which you can see during the life of the child, yet the characteristic lesion is present. There is a croupous inflammation, which may be confined to the larynx, or may involve the tonsils, pharynx, and larynx, but the amount of membrane is so small that it escapes observation. These are the two varieties of malignant cases.

LECTURE XXII.

DIPHTHERIA—CONCLUDED.

Diphtheria (concluded); Symptoms in Ordinary Cases, Prognosis.—Consideration of Individual Symptoms: False Membrane, Discharge of Fluid from Mouth and Nose, Enlargement of the Glands of the Neck, Rigors, Febrile Movement, Laryngeal Symptoms, Vomiting, Diarrhœa, Condition of Pulse, Urine.—Convalescence, Sequelæ, Prognosis, Local and General Treatment.

GENTLEMEN: At the close of our last lecture we finished the consideration of the symptoms of the second set of cases of malignant diphtheria.

Symptoms in Ordinary Cases.—The more ordinary form of the disease, and that which is now seen and has been for years most frequently seen in New York, occurs in this way. The first symptom of which the child or adult complains is a feeling of soreness in the throat. If you look at the throat you will find that there is a general congestion, and upon one of the tonsils, or at some part of the pharynx, a small white patch of false membrane may be seen. At the same time there may or may not be a febrile movement. Sometimes it is not developed until several days after the child complains of sore throat.

This fall, one of the house staff of Bellevue Hospital, who died of this disease, complained of nothing but sore throat, which came on one day after going his usual rounds through the wards. I found, upon looking at his throat, large white patches, and told him that he must go home and go to bed, which he immediately did. There was no febrile movement until the third or fourth day, and he died upon the eighth day.

The false membrane gradually extends over more and more of the throat until, finally, in many cases, all of the throat which you are able to see is covered with a thick layer of false membrane. This is white, at first, but afterward becomes of a gray or dirty brown color. After this there may or may not be swelling of the glands of the neck. There may or may not be disturbances of the stomach. Some of the patients will vomit, while others will complain simply of a feeling of nausea. There is always some obstruction of the throat. The patients feel sick. They are disposed to go to bed and keep quiet, although to this rule there are some exceptions. There are some patients who will remain up and go to business during the first two, three, or four days of the disease. If we examine the urine we will usually find it albuminous, and sometimes it is diminished in amount. The disease seems to progress during a period of from two to seven days. The membrane constantly extends, involving more and more of the throat, and the patient's general condition becomes worse. The febrile movement, if absent at the beginning, will develop on the third, fourth, or fifth day. About the end of the first week, somtimes a day before or a day after, we have to expect that the inflammatory process may extend to the larynx, and that it too will be covered with false membrane. Occasionally, when this happens, the patients become at once a good deal worse. There is difficulty in breathing, and, in addition, the obstruction is more marked. There may be drowsiness, alternating with delirium.

In the cases in which the inflammatory process does not extend to the larynx, we find that at about the end of the first week the disease has run its course, and the false membrane begins to be detached. The detachment of the false membrane is accompanied by an increase in the function of the mucous glands of the parts, so that there is a very profuse flow of mucus. The patient will constantly cough up quantities of mucus, and with this there will be fragments of false membrane. After this the patients usually get better. The throat will gradually become cleared of the false membrane, and by the end of two weeks, sometimes a little longer, convalescence will fairly set in.

In the cases in which the lesion extends to the larynx, however, the condition of the patient is much worse. The difficulty in breathing is pronounced and there may be cerebral symptoms. The condition of the larger number of patients continues to get worse. The breathing becomes more and more difficult, the cerebral symptoms become more marked, and the patient dies at the end of a few hours or days, after the development of the laryngeal symptoms. In other cases. however, the progress of the disease is more favorable. The dyspnœa may continue one, two, three, or four days, and yet the patient succeeds in breathing. After this the false membrane may begin to loosen and the patient commences to cough it up. The dyspnœa gradually diminishes, the cerebral symptoms disappear, and the general condition of the patient is improved ; and again at the end of two weeks the patient may enter upon convalescence.

Less frequently we meet with cases in which the lesion is confined to the larynx. The patient does not complain of sore throat and the symptoms are the same as those of crupous laryngitis. There is a febrile movement, more or less developed, and there are the local laryngeal symptoms consisting of a stridulous cough, breathing, and voice, or loss of voice. The dyspnœa constantly becomes worse. There are developed carbonic acid poisoning and cerebral symptoms, and finally the patient dies.

In other cases, again, the disease runs a very mild course indeed. The patient is not, at any time, very sick; the throat lesions are not very marked; there is a patch of false membrane upon one of the tonsils which does not spread, or there may be a patch upon some part of the mucous membrane of the pharynx, but it will remain confined to a moderate area. The constitutional symptoms are equally slight. There is very little febrile movement, and at the end of five, six, or seven days, the patch becomes loosened and is expectorated, or coughed up, and the patient gets well, sometimes without being confined to the bed.

In still other cases the disease begins in the same mild way. There is only a small patch of false membrane upon some part of the throat and slight constitutional symptoms. In this condition the patient may continue for several days, and thus give strength to the opinion that the case is to be a mild one. But after this the disease suddenly becomes worse, the principal change being in the constitutional symptoms. Suddenly there will be very decided obstruction to the breathing, with or without the development of a febrile movement. The throat will be more and more involved by false membrane, which is especially apt to extend to the larynx. When this happens, the patient will develop laryngeal symptoms and become very ill indeed. Most frequently these cases end in death, although they begin in a mild way.

In all of the different varieties of the disease there always exists the danger of the sudden death of the patient from syncope. The danger is especially great in the severe cases, just at the time when convalescence is beginning. It is also true in the mild cases where the patients are not confined to the bed.

Individual Symptoms.—We now come to the consideration of the individual symptoms. In the first place we shall speak of *false membrane.* The membrane appears first upon one of the tonsils or upon some part of the pharynx or posterior nares, or in the larynx, and then, in the majority of cases, extends to involve the adjacent parts. In some cases, especially in malignant ones, there is an escape of *dirty, brownish fluid* from the mouth or nose, and there may also be *bleeding* from the mouth, nose, or gums. The *glands of the neck,* either the lymphatic or salivary glands, are swollen. The degree of swelling varies very much in different cases. Sometimes the swelling is succeeded by a suppurative inflammation. *Rigors* marking the invasion of the disease in a moderate number of cases. The *febrile* *movement* may be absent during the whole course of the disease, or it may be present at the commencement of it and continue nearly all the time, until the patient's death; or it may be present at first and afterward subside, or absent at first and appear several days after the development of the throat difficulty. The *symptoms referable to the larynx* are only present when it is involved in the inflammatory process. Under such circumstances there are the regular symptoms of croupous laryngitis. There is a laryngeal voice, stridulous breathing, stridulous cough, and dyspnœa due to obstruction of the larynx. In the larger number of cases the obstruction is so great as to prove fatal. In other cases it is more moderate, the patients continuing to live until the inflammatory process has run its course and subsides. *Vomiting* is at times present in the early part of the disease, or it may not occur until late, and in some cases there is no vomiting. But in these latter cases there is some nausea, and it is easy to cause the patients to vomit. In a few cases there is *looseness of the bowels*, usually corresponding to the febrile movement. If at the beginning of the disease the fever is pronounced, the *pulse* will be moderately rapid and full. When there is no febrile movement, the pulse is feebler and more rapid than normal. In all the fatal cases it becomes rapid and feeble as death approaches. The *urine* is, as a rule, albuminous and diminished in quantity. The albumen may continue to be present for some time after the recovery of the patient.

Convalescence.—Convalescence is often prolonged after the disease proper has run its course. Although the patients are not very much prostrated, they are feeble, anæmic, have no appetite, and may continue in this condition for weeks or months. It is at the commencement of convalescence that we have to expect the possible occurrence of death from syncope, and this is a point which it is important to remember. It is important to keep the patients quiet at this time, not allowing them to get up too soon and go about. They should be kept quiet in bed for a certain length of time after convalescence has begun.

Sequelæ.—There are certain regular sequelæ of diphtheria. In some cases there is paralysis of some of the voluntary muscles after the patients get well. The muscles which are paralyzed most frequently are those of the soft palate, pharynx, and larynx. Next in frequency are those of the eye, and in this locality there may be an involuntary muscle paralyzed. The ciliary muscle or the muscle of the iris may become paralyzed, the pupil being dilated and the patient unable to see near objects with any certainty. Besides these muscles, there is frequently paralysis of the muscles of the arms, legs, face, and trunk. These paralyses are, as a rule, only temporary, lasting for perhaps weeks and months, after which time they gradually disappear. Less frequently, however, such a paralysis may be permanent.

Prognosis.—The prognosis of diphtheria depends somewhat upon the particular epidemic which is prevailing, somewhat upon the age of the patient, and whether or not the larynx is involved by the disease.

There are some epidemics in which the malignant form prevails to a great extent and the larger number of the patients die. There are other epidemics in which the malignant cases are much less frequent and the prognosis is better. Generally speaking, there are more malignant cases during the increase of an epidemic than when it is declining. The younger the patient the worse is the prognosis. When the larynx is involved, the prognosis is always much worse than when it is not affected. There are no cases of diphtheria in which the prognosis is not doubtful. The disease is at best a very dangerous one.

Treatment.—In the treatment of this disease there are to be considered local and general measures.

General Measures.—In the general treatment of the disease there should be administered such drugs as will act upon the general system. Alcohol is the favorite with many, and may be in the form of wine or liquor. When the alcoholic treatment of diphtheria is spoken of, it is understood that alcohol is given not merely for the purpose of sustaining the patient, but, in addition, for its constitutional effect. With this end in view it is given in large and repeated doses, and usually in a concentrated form, as brandy or whiskey. For an adult, a half ounce or an ounce of one of these is given every hour or half hour; to a child, a drachm or half a drachm of brandy or whiskey is given every hour or every two hours. It is given in large quantities in this way with the idea of getting the physiological effect of alcohol upon the body, and this you observe is a very different thing from giving stimulants when the patient's strength begins to diminish. This plan of treatment is one which is a favorite with many practitioners, and it has seemed to be successful in some places. Besides using alcohol in this way, it may be used simply as a cardiac stimulant after the disease has lasted for some time and after the heart's action begins to flag.

The use of the tincture of the chloride of iron, by itself or alternately with chlorate of potassium, is another favorite plan of treatment. It has been used very extensively in this city. The ordinary plan is to have two solutions made, one with the muriated tincture of iron and the other with the chlorate of potash. Of the iron, five to twenty drops are given according to the age of the patient; of the potassium, thirty to sixty grains are given in like manner. These drugs are given alternately every hour or two.

Another plan of treatment is to give quinine in very considerable doses. Still another plan which is not in frequent use, consists in the administration of calomel up to the point of salivation. Some physicians give cubebs in large doses, either in the form of powders, or as a tincture. The hyposulphate of soda, in doses of one to ten grains every three or four hours, has also been used. Carbolic acid and salicylic acid have also been given internally in small doses. The benzoate of soda has been very much spoken of as an internal remedy within the last year or so, in doses of twenty grains to a drachm every two or three hours. Emetics are sometimes given to relieve the laryngeal dyspnœa. The three plans of treatment which are most frequently used at the present time are the administration of alcohol in large doses, the use of the tincture of iron and chlorate of potassium in frequently repeated doses, and the use of benzoate of soda in large and repeated doses.

Local Measures.—As regards the local treatment, there are again a number of substances which have been employed. These have been applied locally either by means of a brush, spray, or by gargling; most commonly, either with a brush or spray, for the patients cannot gargle very well. Of inhalations, the vapor of hot water and that produced by the slacking of lime have been used. Chloride of iron may be applied with a brush or spray to the inflamed parts. The nitrate of silver has been used, but is not now so much in vogue as formerly. The strong mineral acids were at one time a good deal used, but are not so much used of late. Alum, the sulphate of iron and burnt chloral hydrate, have all been used as local applications. The topical applications most frequently used are solutions of carbolic acid, salicylic acid, tannic acid and the chloride of iron. If they are to be of any service, they must be applied very frequently, at least every hour or two according as the patients will endure it.

In those cases in which the larynx is involved, the question of tracheotomy always comes up, and it is always a difficult question to answer. As a matter of experience, the operation does not usually save the life of the patient, and yet, in any given case, when a patient is apparently dying simply from suffocation, it is very difficult for one to decide to let the case go on without performing an operation, even though experience has shown that it is almost useless. So that, as a matter of fact, tracheotomy is often performed in cases where the larynx is involved, but, it must be confessed that the results have usually been unsuccessful.

LECTURE XXIII.

INFLAMMATIONS OF THE PLEURA.

Inflammations of the Pleura.—Anatomy of the Pleura.—Varieties of Pleuritic Inflammation.—General Pathology.—Duration of Inflammatory Process.—Dry or Acute Pleurisy; Pathology, Etiology, Symptoms, Physical Signs, Prognosis, Treatment.—Diaphragmatic and Mediastinal Pleurisies; Symptoms, Treatment.

GENTLEMEN:—We begin to-day the study of the inflammations of the pleura.

Anatomy.—The pleura, as you know, is described by anatomists as being one of the serous membranes, and, as a closed sac, having a parietal and visceral layer. For our purpose, however, it is necessary to think of the pleura somewhat differently. There is in the upper part of the body the thoracic cavity, in which are contained the lungs and heart. The inner surface of this cavity is lined with a connective-tissue membrane, which is usually called the costal pleura, or diaphragmatic pleura, according as it covers the inside of the ribs or the inside of the diaphragm. The thoracic cavity is divided into two lateral halves, the right and left, the division being effected by another connective-tissue membrane, called the mediastinal pleura. The lungs are covered by connective-tissue membrane called the pulmonary pleura, which, in fact, is only a part of the lungs. The lungs are composed, as you know, of veins, bronchi, air-vesicles, blood-vessels and connective-tissue, the pulmonary pleura being a part of the connective tissue of the lungs, and hence, inflammations of the pulmonary pleura belong to those of the lungs. In the same way the costal pleura is part of the chest-wall. It is a connective-tissue membrane lining the chest-wall, and is continuous with the tissues beneath, so that it really forms a part of it. The mediastinal pleura again has a somewhat different anatomy from the pulmonary or costal pleura. It divides the thoracic cavity into two lateral halves. When we speak of idiopathic pleurisy, the particular parts which we think of as being affected by a primary inflammation are the costal, the diaphragmatic, and the mediastinal pleuras. We do not, as a general thing, mean that the pulmonary pleura is affected. If we look at the inner surface of the costal pleura, we find that it is lined with a single continuous layer of flat, nucleated, endothelial cells. Beneath this the pleura is formed of connective tissue, the fibres of which run parallel to each other; and at their edges are fixed connective-tissue cells. The texture of the pleura is further reinforced by yellow, elastic fibres. Embedded in this are blood-vessels in moderate numbers, nerves, and lymphatic vessels in very large numbers. There are large and small trunks, and connecting these are irregular, stellate spaces. You will observe that the connective tissue, instead of being solid, is really a spongy tissue; it is honey-combed. This extensive lymphatic system of the pleura is of considerable importance in relation to inflammations of the pleura, for it is by means of this that the products of inflammation are taken up and removed from the pleural cavity and the surface of the pleura.

Varieties of Inflammation.—The pleura may be inflamed in several different ways, and hence there are varieties of pleurisy according to the products of inflammation. First, there is pleurisy with the production of only fibrin; second, pleurisy with the production of fibrin and serum; third, pleurisy with the production of fibrin, serum, and pus; and fourth, pleurisy with the production of new connective tissue, or pleurisy with adhesions, as it is called.

General Pathology.—The pathological process in pleurisy with the production of only fibrin, and pleurisy with the production of both fibrin and serum, is essentially the same. The first stage of the inflammatory process is marked by the blood-vessels containing an increased amount of blood and a falling off of a certain number of endothelial cells from the free surface of the pleura; in other words, there is a congestion with desquamation of endothelial cells. The next thing which is noticed is that the pleura loses its natural glistening and shining appearance. It looks dull and may even appear a little rough. When examined under the microscope, it is found that this change is due to the presence of a certain amount of coagulated fibrin upon the surface of the pleura. In the early stages the fibrin is present only in very small amount, it being scattered here and there in little knots or threads upon the surface of the pleura. At the same time there is some swelling of the fixed connective-tissue cells, and they are increased in number. Pus-cells are also found in the substance of the pleura, although these are not numerous. This stage corresponds to the first forty-eight hours of the disease.

The next step in the inflammatory process is for the fibrin on the free surface of the pleura to become increased in amount. The extent to which the fibrin is produced varies in different cases with the severity of the inflammatory process, but there is commonly enough fibrin to form a continuous layer over the free surface of the pleura. If serum is produced at the same time, we find this in the cavity of the pleura. The production of connective tissue-cells and pus-cells continues, but still it is not excessive, being principally confined to the inner layers of the pleura, or those parts nearest the lungs.

The next step in the inflammatory process is that cells begin to make their appearance in the layer of fibrin. Some of these cells are cuneiform in shape; some are rounded with two or more nuclei, and others are polygonal in form. It seems probable that these cells are derived from the connective-tissue cells in the pleura itself, and that they are formed in the pleura and then find their way into the layer of fibrin which coats the inner surface of the pleura. As time elapses these cells become more and more numerous and the fibrin begins to be absorbed, and, by the fourth or fifth day of the inflammatory process, new blood-vessels have been formed, and it is found that what was at first a simple layer of fibrin is now a layer composed of cuneiform cells, among which are new blood-vessels, which become continuous with the original blood-vessels of the costal pleura. After the disease has lasted four or five days, if the pleural blood-vessels are artificially injected, it will be found that the fluid will pass into the new blood-vessels formed in the layer upon the inner surface of the pleura. Subsequently, the regular progress of the pleurisy is for the fibrin to gradually diminish in amount until there is no longer any left. The connective-tissue cells also undergo a change. A certain number of them undergo fatty degeneration and disappear. The others, however, remain, and basement-substance is formed. As time elapses the cells become less and less numerous and the basement-substance more and more abundant. The cells also begin to resemble the original cells of the pleura, and finally, as a result of this process, there is formed upon the inner surface of the pleura a layer of permanent new connective tissue. The fibrin, which is produced in the first place, is not confined to the costal pleura, a certain amount also being produced upon the pulmonary pleura; and as these two pleuras lie pretty close together, it is found that there are adhesions taking place between them which are composed of fibrin and of permanent new connective tissue. It is important to remember the natural termination of these cases of pleurisy. The tissue does not return to its normal condition, but upon the free surface there is a layer of new connective tissue, and between the pulmonary and costal pleuras there is a certain number of permanent adhesions. When the patient has once suffered from this form of pleurisy, he will always have a permanent lesion, and there will be a larger or smaller number of adhesions between the pulmonary and costal pleuras.

Duration of Inflammatory Process.—The time during which this inflammatory process is running its course varies. The fibrin and serum begin to be formed within three or four hours, and by the end of twenty-four or forty-eight hours there is always a considerable production of fibrin. After this, however, the fibrin may remain unabsorbed for a long time. Occasionally the process runs its course in a couple of weeks. In other cases, however, the fibrin and serum will persist for weeks or months without being absorbed, and when this happens the production of new connective tissue is retarded, and is not effected until the fibrin and serum are absorbed.

Dry or Acute Pleurisy.—We come now to pleurisy with the production of only fibrin. This form is very often called dry pleurisy, because there is no serum, or, at all events, very little. It is also called acute pleurisy, because it runs an acute course.

Pathology.—The lesion consists in the inflammation of some part of the pleura, and upon this inflamed part is produced a layer of fibrin, following which are the changes I have already described. The part of the pleura that is inflamed is commonly circumscribed. It does not involve the whole of one side of the pleura, but only a small part of the costal, mediastinal, or diaphragmatic pleuras. As a pretty rare exception, the whole of the costal pleura upon one side is involved.

Etiology.—The causes of this kind of inflammation are: first, exposure to cold; second, penetrating wounds of the wall of the thorax; and third, fractures of the ribs, if the broken ends of the rib penetrate into the pleura. It is also found as a complication of some diseases, as pyæmia, puerperal fever, peritonitis, and Bright's disease. There is also a strong predisposition in some individuals to this disease, and they suffer from repeated attacks of it. Occasionally a patient will have an attack during several succeeding winters.

Symptoms.—The evidences of dry pleurisy are partly rational symptoms and partly physical signs. The rational symptoms vary very much in their intensity in different cases. When present, they consist of a febrile movement, of pain in the side, of a short, dry cough, and of a certain amount of dyspnœa. In some cases these symptoms are well marked. The temperature runs up to 102°, 103°, or 104°. The patients have a full and somewhat rapid pulse, and a pain in the side, which may be very pronounced. The cough is distressing. The dyspnœa is well marked, the patient breathing rapidly and with difficulty. In other cases these rational symptoms are almost wanting. The febrile movement is very slight indeed, the pain in the side is comparatively moderate, the cough is slight, and the breathing comparatively easy. Some of these patients will walk about, simply complaining of a pain in their side. By placing your ear to the chest, you will find the characteristic signs. Other patients have a febrile movemen, which is sufficient to keep them in the house or in bed. They will tell you that they have a little pain, but that they do not feel very sick. In other cases the pain will be almost the only symptom, and it will be quite severe.

Physical Signs.—The characteristic physical sign of dry pleurisy is what is called the friction-sound. This is heard directly over that part of the pleura which is inflamed. The sound is not always of the same character; sometimes it is of a peculiar rubbing sound, as if two surfaces were being rubbed against each other. At other times it resembles the sound ordinarily called *crepitant* râle, that is, a dry, fine crackle, which is heard directly under the ear. In other cases it is characterized by what is called sub-crepitant râle, that is, a rather coarser and little moister râle, heard directly under the ear. These are the three varieties of friction-sounds. The sound seems in all cases to be due to the same cause, namely, a rubbing together of the roughened costal and pulmonary pleuras. This crepitant râle is not, however, always present. Even while the pleurisy is in progress, the chest may be auscultated at different times during the day without hearing this sound, and again it will be present. If you do not hear it, it is well to make the patient cough or take a deep inspiration. In this way you may be able to hear a friction-sound which was previously inaudible. Still, even this means will not always develop it. This physical sign is not a constant one; it comes and goes, and, more than this, you cannot expect to hear it except it be due to inflammation of the costal pleura. If it is the mediastinal or diaphragmatic pleura which is inflamed, of course you cannot hear the friction-sound, for you cannot get your ear near enough to the inflamed pleura.

Prognosis.—The duration of the disease is only for a few days—about a week, or sometimes two. Its natural termination is in the recovery of the patient. Still the disease is by no means a harmless one, for the patients get well with a thickened pleura and a certain number of adhesions between the pulmonary and costal pleuras. And besides this, there is a predisposition to other attacks. Each attack of dry pleurisy which patients have leaves them with an additional amount of pleuritic thickening and an additional number of adhesions between the pulmonary and costal pleuras. It is for this reason that, after patients have had a number of attacks of this form of pleurisy, the lesion will finally affect their health in a serious manner. As a result of this, in some cases there is a development of interstitial pneumonia, empyema, or bronchitis. There are, however, a few exceptions to this ordinary course of the disease. Very seldom the rational symptoms are decidedly marked. The patients seem to be very ill, having a fever, pain, cough, and dyspnœa. Added to this there may be, in some cases, extreme prostration, and the patients, instead of recovering, die of the disease. After death it is found that the cause of the marked rational symptoms and of death was the enormous production of fibrin. The fibrin, instead of forming a layer which simply coats the pleura, forms a mass that compresses the lung, just as if the pleural cavity was filled with serum. Such cases are fortunately very rare. When they do occur, they prove fatal, and generally within a few days.

Treatment.—The indications for treatment in ordinary cases of dry pleurisy are twofold. They are, in the first place, to diminish the severity of the inflammatory process, and, in the second place, to alleviate the pain from which the patients suffer. To reduce the intensity of the inflammation, cups or counter-irritation may be applied. If the patient is strong and robust, I think there is considerable advantage in applying wet cups and taking a certain amount of blood from the wall of the thorax. This procedure is usually an agreeable one to the patients, for they feel better in a very short time after the local abstraction of blood. If, however, the patient is not particularly strong and robust, dry cups or counter-irritation may be applied. If the latter is used, the best way of applying it is in the form of a blister, as it is desirable to have the counter-irritation rapid and decided. To relieve the pain, the best remedy is opium in moderate doses, given either by the mouth or by means of a hypodermic syringe. The opium does not usually require to be given to any very great extent, but simply in sufficient amount to keep the patient tolerably comfortable. In the majority of cases this constitutes all the treatment that will be required.

Diaphragmatic and Mediastinal Pleurisies.—When the inflammatory process is located in the diaphragmatic or mediastinal pleura, very little can be done in the way of counter-irritation, for you cannot apply the blister over the inflamed pleura. In these cases, the pain is often particularly severe. If it is the mediastinal pleura that is affected, the pain is usually referred to the sternum, and this may be mistaken for a case of pericarditis. If the lesion is confined to the diaphragmatic pleura, the pain is referred to the thorax, or to the upper part of the abdominal cavity or lower down, even as far as the hips. It is possible to mistake these cases for those of peritonitis. In the cases of mediastinal and diaphragmatic pleurisy, the disease runs the same course as in the costal, except the existence of pain. All that you have to do in these cases is to administer opium in such doses as will keep the patient comfortable.

LECTURE XXIV.

PLEURISY WITH EFFUSION.

Subacute Pleurisy, or Pleurisy with Effusion; Pathology, Etiology, Rational Symptoms, Ordinary and Exceptional Physical Signs, Course and Termination, Diagnosis, Treatment.

SUBACUTE PLEURISY, OR PLEURISY WITH EFFUSION.—*Gentlemen:* The next form of pleurisy which we have to consider is that with the production of fibrin and serum in a considerable amount. This form of pleurisy is very commonly called pleurisy with effusion; it is also sometimes called subacute pleurisy. It is the commonest form of pleurisy.

Pathology.—The inflammatory process involves, as a rule, the whole of the pleura upon one side of the chest, beginning at the costal pleura, and there reaching its greatest intensity, but involving also the diaphragmatic, mediastinal, and pulmonary pleuras upon the same side of the chest. If the patient dies from this disease, we find, upon examination, the entire pleura coated with a layer of fibrin, the thickness of which varies with the intensity of the inflammatory process. It is thickest, as a rule, over the costal pleura. Besides this, in some cases, there are fibrous adhesions between the pulmonary and costal pleuras. If this layer of fibrin be stripped off, it is found that the pleura is not very much thickened, the apparent thickness being due not to a change in the tissue, but to the layer of fibrin spread over its surface. If the patient is convalescent at the time of death, we find, mixed with the fibrin, new connective-tissue cells with or without basement-substance. If the patient recovers and finally dies of some other disease, we find that the fibrin has entirely disappeared, and there has been left a layer of new connective tissue upon the surface of the pleura, together with adhesions between the pulmonary and costal pleuras.

Besides changes upon the surface of the pleura, there is found serum in the pleural cavity, which varies in amount. At times it is so abundant that the entire pleural cavity is filled and distended, the diaphragm being pushed down by it. In other cases the pleural cavity is production of new connective tissue is retarded, and is not effecte until the fibrin and serum are absorbed.

DRY OR ACUTE PLEURISY.—We come now to pleurisy with th production of only fibrin. This form is very often called dry pleurisy, because there is no serum, or, at all events, very little. It is also called acute pleurisy, because it runs an acute course.

Pathology.—The lesion consists in the inflammation of some part of the pleura, and upon this inflamed part is produced a layer of fibrin, following which are the changes I have already described. The part of the pleura that is inflamed is commonly circumscribed. It does not involve the whole of one side of the pleura, but only a small part of the costal, mediastinal, or diaphragmatic pleuras. As a pretty rare exception, the whole of the costal pleura upon one side is involved.

Etiology.—The causes of this kind of inflammation are: first, exposure to cold; second, penetrating wounds of the wall of the thorax; and third, fractures of the ribs, if the broken ends of the rib penetrate into the pleura. It is also found as a complication of some diseases, as pyæmia, puerperal fever, peritonitis, and Bright's disease. There is also a strong predisposition in some individuals to this disease, and they suffer from repeated attacks of it. Occasionally a patient will have an attack during several succeeding winters.

Symptoms.—The evidences of dry pleurisy are partly rational symptoms and partly physical signs. The rational symptoms vary very much in their intensity in different cases. When present, they consist of a febrile movement, of pain in the side, of a short, dry cough, and of a certain amount of dyspnœa. In some cases these symptoms are well marked. The temperature runs up to 102°, 103°, or 104°. The patients have a full and somewhat rapid pulse, and a pain in the side, which may be very pronounced. The cough is distressing. The dyspnœa is well marked, the patient breathing rapidly and with difficulty. In other cases these rational symptoms are almost wanting. The febrile movement is very slight indeed, the pain in the side is comparatively moderate, the cough is slight, and the breathing comparatively easy. Some of these patients will walk about, simply complaining of a pain in their side. By placing your ear to the chest, you will find the characteristic signs. Other patients have a febrile movemen, which is sufficient to keep them in the house or in bed. They will tell you that they have a little pain, but that they do not feel very sick. In other cases the pain will be almost the only symptom, and it will be quite severe.

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Prognosis.—The duration of the disease is only for a few days—about a week, or sometimes two. Its natural termination is in the recovery of the patient. Still the disease is by no means a harmless one, for the patients get well with a thickened pleura and a certain number of adhesions between the pulmonary and costal pleuras. And besides this, there is a predisposition to other attacks. Each attack of dry pleurisy which patients have leaves them with an additional amount of pleuritic thickening and an additional number of adhesions between the pulmonary and costal pleuras. It is for this reason that, after patients have had a number of attacks of this form of pleurisy, the lesion will finally affect their health in a serious manner. As a result of this, in some cases there is a development of interstitial pneumonia, empyema, or bronchitis. There are, however, a few exceptions to this ordinary course of the disease. Very seldom the rational symptoms are decidedly marked. The patients seem to be very ill, having a fever, pain, cough, and dyspnœa. Added to this there may be, in some cases, extreme prostration, and the patients, instead of recovering, die of the disease. After death it is found that the cause of the marked rational symptoms and of death was the enormous production of fibrin. The fibrin, instead of forming a layer which simply coats the pleura, forms a mass that compresses the lung, just as if the pleural cavity was filled with serum. Such cases are fortunately very rare. When they do occur, they prove fatal, and generally within a few days.

Treatment.—The indications for treatment in ordinary cases of dry pleurisy are twofold. They are, in the first place, to diminish the severity of the inflammatory process, and, in the second place, to alleviate the pain from which the patients suffer. To reduce the intensity of the inflammation, cups or counter-irritation may be applied. If the patient is strong and robust, I think there is considerable advantage in applying wet cups and taking a certain amount of blood from the wall of the thorax. This procedure is usually an agreeable one to the patients, for they feel better in a very short time afte local abstraction of blood. If, however, the patient is not particu strong and robust, dry cups or counter-irritation may be applied. the latter is used, the best way of applying it is in the form blister, as it is desirable to have the counter-irritation rapid and cided. To relieve the pain, the best remedy is opium in mode doses, given either by the mouth or by means of a hypodermic syri The opium does not usually require to be given to any very great tent, but simply in sufficient amount to keep the patient toler comfortable. In the majority of cases this constitutes all the tr ment that will be required.

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LECTURE XXIV.

PLEURISY WITH EFFUSION.

Subacute Pleurisy, or Pleurisy with Effusion; Pathology, Etiology, Rational Symptoms, Ordinary and Exceptional Physical Signs, Course and Termination, Diagnosis, Treatment.

SUBACUTE PLEURISY, OR PLEURISY WITH EFFUSION.—*Gentlemen:* The next form of pleurisy which we have to consider is that with the production of fibrin and serum in a considerable amount. This form of pleurisy is very commonly called pleurisy with effusion; it is also sometimes called subacute pleurisy. It is the commonest form of pleurisy.

Pathology.—The inflammatory process involves, as a rule, the whole of the pleura upon one side of the chest, beginning at the costal pleura, and there reaching its greatest intensity, but involving also the diaphragmatic, mediastinal, and pulmonary pleuras upon the same side of the chest. If the patient dies from this disease, we find, upon examination, the entire pleura coated with a layer of fibrin, the thickness of which varies with the intensity of the inflammatory process. It is thickest, as a rule, over the costal pleura. Besides this, in some cases, there are fibrous adhesions between the pulmonary and costal pleuras. If this layer of fibrin be stripped off, it is found that the pleura is not very much thickened, the apparent thickness being due not to a change in the tissue, but to the layer of fibrin spread over its surface. If the patient is convalescent at the time of death, we find, mixed with the fibrin, new connective-tissue cells with or without basement-substance. If the patient recovers and finally dies of some other disease, we find that the fibrin has entirely disappeared, and there has been left a layer of new connective tissue upon the surface of the pleura, together with adhesions between the pulmonary and costal pleuras.

Besides changes upon the surface of the pleura, there is found serum in the pleural cavity, which varies in amount. At times it is so abundant that the entire pleural cavity is filled and distended, the diaphragm being pushed down by it. In other cases the pleural cavity is two-thirds or one-half filled with serum. The serum may be clear or turbid, owing to the admixture of a few pus-globules, or flakes of fibrin, or it may contain a certain amount of blood. The lung is compressed by the fluid, and the particular way in which it is compressed varies somewhat in different cases. The rule is that it is compressed toward the vertebral column, the fluid occupying the rest of the pleural cavity. In other cases the lung will be compressed upward, so that, while the upper part of the pleural cavity is occupied by the lung, the lower part is occupied by the fluid. In other cases the lung will occupy the anterior half of the pleural cavity, and the fluid the posterior half. The compression of the lung is also influenced by the existence of adhesions. If the adhesions be very strong, they will hold parts of the lung in contact with the costal pleura, so that it will be irregularly compressed by the fluid.

Etiology.—The causes of this form of pleurisy are the same as those of pleurisy, with the production of only fibrin. Most of the cases that we see are idiopathic, the disease occurring in persons previously in good health. Occasionally there is obtained a distinct account of exposure to cold. At other times there will be a history of a traumatism, as wounds of the wall of the thorax and fractures of the ribs. It also occurs as a complication of pneumonia, phthisis, puerperal fever, peritonitis, and Bright's disease.

Rational Symptoms.—The rational symptoms vary somewhat in different cases, and according to these it is convenient to divide the cases into two classes.

First Set of Cases.—In the first set of cases the invasion of the disease is acute. A patient who has previously been in good health is suddenly attacked with rigors, which are accompanied and followed by a febrile movement of considerable intensity. The pulse is full and rapid. There are pains all over the body, in the bones and muscles, and there may be a headache. There is pain upon the affected side. In some cases there is a cough, and there may be vomiting. At the commencement of the attack the breathing is rapid and shallow. These acute constitutional symptoms last for twenty-four hours; or occasionally seven or eight days, and then subside—sometimes abruptly, at other times gradually. If the products of inflammation are absorbed rapidly, the patient will recover rapidly; or, if these products are slowly absorbed, the patient continues to be sick for a considerable length of time after all acute symptoms have ceased.

The pain which belongs to this form of pleurisy is situated regularly over the inflamed pleura. It is, however, not always situated here, and, owing to this fact, errors in diagnosis may be made. The patients will sometimes refer the pain to a point low in the lumbar region, or even in the hip, and under such circumstances it has been mistaken for that which belongs to nephritic colic. Again, the pain may be characterized as sharp and severe, and be referred to the epigastric region or some point in the abdomen. When this is the case, the disease may be mistaken for peritonitis. Other patients will refer the pain to the opposite side of the thorax. The pain is usually severe, and is increased by inspiration. With the increase in the production of serum, the pain usually diminishes.

Second Set of Cases.—In the second set of cases the invasion of the disease is not acute. It is not ushered in by any pronounced symptoms. In fact, in many cases it is difficult to tell when the disease began, so mild are the symptoms. There is present pain in the affected side, but it is neither severe nor sharp, as in the acute cases. There is also a certain amount of dyspnœa, but this is only manifested when the patient exercises. He will tell you that he breathes well enough except when he walks fast, or goes upstairs, or makes some unusual exertion. Coughing is at times present, and at other times entirely absent. There is a gradual loss of health, a loss of strength, and the patient become somewhat anæmic. Many patients are not confined to bed at all; especially is this true among the laboring classes. You may occasionally find a man who carries a hod, or digs, or does some other severe work, with one of his pleural cavities half or two-thirds filled with serum. Most commonly there is some elevation of temperature, the thermometer indicating 99° or 100°. The temperature, as a rule, is higher in the afternoon than in the morning. Very often the patients are not conscious of the existence of a febrile movement.

Physical Signs.—The physical signs of pleurisy with effusion are the same in the two sets of cases of which we have just spoken. At the very commencement of the disease there is a friction-sound. This is obtained before much serum accumulates in the pleural cavity, but during the existence of the effusion it is absent. It returns, however, at the close of the disease. If the patient gets well, all the serum is absorbed and the lung expands. The friction-sound obtained at the close of the disease is commonly coarse, like the coarse mucous râle. After the fluid is produced, and fills or partly fills the pleural cavity, there are other physical signs which are regularly obtainable. Below the level of the fluid in the chest there is flatness upon percussion. If we put our hand upon the side of the patient and cause him to count, we find that the vocal fremitus is absent. If we put our ear to the side of the chest and listen, we no longer hear the voice or breathing. If we cause the patient to lean forward or backward, this may produce a change in the level of the flatness, owing to a change in the level of the fluid, due to the position of the patient. This change we do not get quite so often as might be expected. Above the level of the fluid, in some cases, there is normal pulmonary resonance, in others exaggerated and higher-pitched pulmonary resonance, and in others there is tympanitic resonance upon percussion. If we listen to the breathing above the level of the fluid, we will find it natural in some cases, in others exaggerated and harsh, and in others it is a bronchial breathing. If we cause the patient to count, in some we find no change in the voice, in others the voice sounds louder and higher-pitched, and in others we obtain a distinct bronchial voice.

At the level of the fluid, dulness is regularly obtained and the peculiar kind of voice which is called egophony, because it is shallower and more like the bleating of a goat than ordinary bronchophony. All these physical signs are usually heard best from behind. Besides this, if the two sides of the chest be measured, it will be found that the side which contains the fluid measures more than the other. In a few cases the intercostal spaces are bulged outward. If the effusion is great, the heart is pushed to one side. This is especially the case if the pleurisy occurs upon the left side of the body. When this happens, the apex of the heart will be pushed over to the right of the sternum, in the fourth or fifth intercostal space.

As the patient gets better and the fluid is absorbed, the line where the voice and breathing can no longer be heard gradually becomes lower. The line of flatness, however, will not go down so rapidly as the line of voice and breathing, which seems to be due to the amount of fibrin that adheres to the surface of the pleura. In some cases there will be a certain amount of dulness left upon the affected side, so that the height of the fluid is measured, not so much by the change in the level of the dulness, as by the change in the level at which the voice and breathing is heard.

Exceptional Physical Signs.—Unfortunately, besides these regular physical signs of pleurisy, there are exceptional cases in which they are changed. There are some cases in which the vocal fremitus persists, even when the chest is half-filled with fluid. Another exception is that bronchial voice and breathing may be heard below the level of the fluid. This is especially the case when there is a great deal of fluid in the pleural cavity. Sometimes there will be a sudden change in the voice and breathing, following aspirations of the chest. In a patient who has one side of his chest filled with serum, there may be bronchial breathing and voice heard over the whole of the affected side. If some of the fluid is drawn off by means of an aspirator, the bronchial breathing and voice will disappear, the physical signs now being those ordinarily obtained. In some cases the voice is heard below the level of the fluid, although the breathing cannot be heard here. By listening below the level of the fluid, sometimes there will be heard cavernous breathing, and gurgles, and yet there is no cavity. These are most likely to be heard in front.

Course and Termination.—The course and termination of the disease varies very much in different cases. A number of patients die suddenly. They will apparently be doing well, although suffering from a certain amount of dyspnœa. They may sit up in bed and fall back dead, or get out of bed to go to the water-closet and fall dead upon the floor. In these cases of sudden death the lung upon the opposite side of the body is sometimes found very much congested, which is given as the reason for the sudden death of the patient. In other cases no anatomical lesion is found to account for it. There will be the ordinary lesions of pleurisy upon one side, and the other lung will be in good condition. It is well to remember that such a termination of pleurisy with effusion is not so very uncommon, and for this reason it is important not to be too sanguine in your prognosis. Even if the patient is doing well you should always remember to let the friends know that he is suffering from a serious disease. If you see the patient in the morning and tell his friends that he is doing well, and in the afternoon he dies, your credit with the family as regards prognosis will not be very good.

There are other cases which last only a few weeks. By the end of this time the serum and fibrin are absorbed, the lung expands, and the patient returns to a condition of health. There are other cases which continue for a much lónger period, from a few months to over a year, at the end of which time the serum and fibrin will be absorbed, the lung will expand, and the patient will return to a natural condition. In other cases, after a time, pus is produced, changes taking place in the tissue of the pleura, and the patient begins to suffer from empyema instead of pleurisy with effusion.

When patients recover they always do so with a certain amount of thickening of the pleura, and usually with adhesions between the pulmonary and costal pleuras. If these adhesions are not very extensive, they do not give rise to very much trouble; but still, a patient who has suffered from pleurisy with effusion will usually be conscious of a difference between the two sides of the thorax for months or years after the attack. If he gets a slight cold or exerts himself too much, he will suffer from pain and discomfort upon the side of the thorax where the pleurisy existed.

If the adhesions are extensive and the thickening of the pleura is very marked, not permitting the lung to expand as it should, there is retraction of the affected side following the attack. When the patient breathes it may often be seen that the intercostal spaces are drawn in by the act of inspiration, instead of being pushed out as in health. When this occurs, there is permanent dulness over the affected side, and in a certain number of patients the breathing and voice are peculiar, and resemble bronchial breathing and voice, but are of a lower pitch.

When fluid is present in considerable amount, and especially when situated upon the left side of the thorax, it is expected that the heart will be pushed toward the right side. The heart may, of course, be pushed aside if the fluid is upon the right side, but the displacement is more marked if the fluid is upon the left side. In some patients, when recovery takes place, the heart returns to its natural position. In others it is held by the adhesions in an abnormal position after the fluid has been absorbed.

In those cases where there are extensive adhesions and pleuritic thickenings, not infrequently the patients go on to suffer from interstitial pneumonia. At other times they suffer from chronic bronchitis or emphysema. In other cases the bronchi become dilated, and in still other cases the patients develop ordinary chronic phthisis. You will observe, then, even in the cases that recover from pleurisy, that the consequences may be very serious.

Diagnosis.—The diagnosis of this condition is sometimes very easy and sometimes very difficult. The diseases with which it is most usually confounded are pneumonia, phthisis, and tumors of the thoracic cavity. The cases of pleurisy which are most apt to be mistaken for pneumonia are those in which the invasion of the disease is acute. Under such circumstances the rational symptoms may be identical with those of ordinary lobar pneumonia, and the physical signs in both diseases may resemble each other to a very considerable extent. In some cases the diagnosis cannot be made out except by the introduction of a hypodermic needle into the pleural cavity, for the purpose of ascertaining whether there is any serum contained in it.

The cases which are likely to be confounded with phthisis are those which have an insidious course, for in these the rational symptoms may be the same as those of phthisis. In phthisis there is a gradual loss of flesh and strength, pain in the side, cough, and a febrile movement, more marked at night. Here again, in some cases, the physical signs may or may not be sufficient evidence for a diagnosis. If they are not sufficiently distinct, it is necessary to resort to the use of the hypodermic syringe to determine whether or not there is serum in the pleural cavity. In the use of the hypodermic syringe for this purpose it is necessary to adopt certain precautions. If the instrument used be an ordinary one with a fine point, it may be introduced into a pleural cavity which contains serum, and yet no serum will pass into the barrel of the syringe on account of the thick coat of fibrin upon the inner surface of the pleura. Another difficulty with the ordinary hypodermic syringe is, that the barrel is so small that the suction produced by withdrawing the piston is not sufficient to draw the fluid into the barrel of the syringe. Hence, by the use of a larger syringe and needle than the ordinary size, better results will be obtained.

Treatment.—In those cases in which the invasion of the disease is acute the treatment varies according to rational symptoms. If the patient is strong and robust, as a rule, considerable relief may be afforded by applying wet cups over the affected side of the thorax. If the patient is not strong and vigorous, dry cups or blisters may be used. To relieve the febrile movement, aconite in small doses may be given. Liquor ammonii acetatis, or spirits of Mindererus, as it is called, may be given, and usually it is necessary to administer opium to quiet the restlessness from which these patients suffer. After the acute symptoms have subsided the question of getting rid of the fluid in the thoracic cavity comes up.

In those cases in which the invasion is insidious and the constitutional symptoms are not well defined, there are two indications to be met. In the first place the fluid should be removed from the pleural cavity, and in the second place the nutrition of the patient must be improved. The exact way in which these cases of subacute pleurisy are to be treated depends on the general condition of the patient. If his nutrition has not suffered up to the time he comes under treatment, your attention should be principally directed to the removal of the fluid. To accomplish this, persistent irritation over the chest by means of blisters must be resorted to, together with the administration of internal remedies, such as iodide and acetate of potassium. If, however, when the patient comes under observation, his nutrition is impaired, he is emaciated and anæmic, less attention should be paid to the fluid in the pleural cavity. The principal efforts should be directed to the improvement of his general health. Such a patient will not bear blisters, nor can his condition be improved by the use of diuretics. Iron, quinine, and the mineral acids should be administered, together with cod-liver oil and cream, if the patient can digest them. It would be well to send him to a warm climate for the purpose of improving his general health. Besides these, the question of the mechanical removal of the fluid from the pleural cavity always presents itself. In regard to the advisability of aspirating the thorax, there is still a considerable difference of opinion in the profession. There may be some cases in which all are agreed that it is advisable to perform this operation. These are the cases in which the dyspnœa is very great and threatens the life of the patient. If the fluid continues in the pleural cavity for any length of time, and all treatment fails to remove it, while the patient's general health is constantly depreciating, all would agree that it was advisable to perform the operation; but opinions differ as to its expediency in the ordinary cases. The reasons given for not aspirating are that in a considerable number of cases a single aspiration is of very little service, for, after the fluid has been drawn off, in the course of a few days it will reaccumulate, and a second aspiration is necessary. This being performed, the fluid again accumulates. and after two or three operations it is found that the fluid changes in character. From being simply serous at first, it has now become purulent; in other words, the case has changed from one of pleurisy with effusion to one of empyema. Another danger from aspiration is that the operation may prove fatal, death taking place suddenly or within twenty-four hours. It is difficult to tell what has been the cause of death under such circumstances, as no lesions are found to account for it. But a considerable number of cases have been recorded in which death has followed so rapidly upon aspiration of the chest that it is necessary to regard the operation as the cause of it.

LECTURE XXV.

EMPYEMA.

Empyema ; Pathology, Invasion, Symptoms, Physical Signs, Prognosis, Treatment.

PLEURISY WITH THE PRODUCTION OF FIBRIN, SERUM, AND PUS.—*Gentlemen:* We come now to the consideration of pleurisy with the production of fibrin, serum, and pus, or empyema, as it is usually called. This form of inflammation of the pleura is commonly confined to one side of the chest, and involves the whole of the pleura on the affected side.

Pathology.—The lesion is most marked in the costal, diaphragmatic and mediastinal pleuras, involving the pulmonary pleura to a less extent. The examination of the chest of a patient who has died from empyema, will show the pleural cavity to contain purulent serum. This varies very much in its consistency in different cases, being at times quite thin and principally composed of serum, with only a moderate number of pus-globules in suspension. In other cases it is very thick, so that it will not run through the needle of an aspirator, unless one of very large size is used. Between these two extremes there are many variations in the consistency of the fluid. This purulent fluid usually accumulates in the lower part of the pleural cavity. Occasionally, however, it occupies its posterior half, while the lung occupies the anterior half. Again, it may be confined to some part of the pleural cavity, owing to adhesions which have taken place. The pleura is commonly coated with a thick layer of fibrin. If, however, the case has lasted for some time, there may be only a very small amount of fibrin upon the surface of the pleura, or it may be altogether absent. The pleura is very much thickened, not by a layer of fibrin, but by a new growth of connective-tissue cells and basement-substance upon its free surface. The proportion existing between the basement-substance and cells varies in different cases.

In some cases the inflammatory process is not confined to the pleura, but extends from the costal pleura to the connective tissue beneath. It may extend to the periosteum, and the perichondrium of the ribs, leading sometimes to necrosis of the ribs and cartilages, or to thickening of the same. It may extend to the fasciæ over the muscles, and beyond this into the muscular tissue and walls outside of the ribs, and even to the skin. The inflammatory process is accompanied either with the production of new connective tissue, or it is a suppurative inflammation with the production of pus. In some cases that have lasted a long time there will be a deposit of the salts of lime, and the thickened pleura will become calcified. In other cases the purulent fluid in the cavity and the fibrin upon the wall of the pleura will become gangrenous; there will be putrefaction with the production of bacteria and the evolution of gas. You will observe the difference between the lesions of empyema and those of pleurisy with effusion, or pleurisy with the production of fibrin and serum. These differences in the lesions lead to great differences in the final results. The natural tendency of pleurisy with effusion is toward recovery, the pleura returning to its normal condition. In empyema as it occurs in adults, the tendency of the disease is not toward convalescence; the pleura has no tendency to return to a normal condition. In the few patients who have empyema and recover, the pleura never returns to a natural condition, but resembles cicatritial tissue.

Invasion.—Empyema begins in several different ways. In the first place, the inflammation may be very active and there will be at once a production of fibrin, serum, and pus. In other words, the case will be one of empyema from the outset. In many cases the disease may begin as a simple pleurisy with effusion, and, after continuing for a variable length of time, it will either gradually or suddenly change into empyema; that is, the inflammatory process will either gradually or suddenly change in character, and pus will be added to the products of inflammation and the tissue itself will undergo a change. Sometimes empyema occurs as a secondary disease, due to abscesses in the wall of the thorax which perforate the pleural cavity, or it may be secondary to abscesses in the liver which perforate the diaphragm, or to abscesses in the abdomen which rupture through the diaphragm into the pleural cavity. In still other cases abscesses of the lungs, or cavities formed in the lungs as a result of chronic phthisis, rupture into the pleural cavity and set up an empyema. In still other cases, empyema may occur as a complication of pyæmia.

Symptoms.—The rational symptoms of empyema will vary with the particular way in which the disease is developed. First, as to the symptoms of those cases in which the inflammation is, from the outset, accompanied with the production of fibrin, serum, and pus. In these cases the invasion of the disease is acute. It is usually ushered in with rigors, which are followed by a pronounced febrile movement. The pulse is full and rapid. There is considerable pain upon the affected side, and decided prostration. In other words, the symptoms are the same as those of acute pleurisy with effusion, but, generally speaking, they are more severe. The prostration is greater, and the patients look very sick at the beginning of the disease. These acute symptoms last for a week or two, at the end of which time the patient may die. In other cases the symptoms behave more like those of pleurisy with effusion. They gradually subside at the end of a week or two, and the disease becomes chronic.

In those cases in which the inflammatory products become gangrenous, the prostration of the patients is excessive. They look very much like those suffering from acute peritonitis. They emaciate very rapidly, the face has a peculiar drawn and haggard appearance, there is apt to be delirium, and they appear to be very ill indeed. The putrefaction of the inflammatory products occasionally takes place early in the disease, or it may not take place until later; but, whether early or late, it gives rise to the same decided symptoms of prostration.

In those cases in which the disease is simply one of pleurisy with effusion, and then gradually changes into empyema, there are, at first, the ordinary symptoms of pleurisy with effusion, and afterward there are added a more marked febrile movement of a hectic character, and a more rapid loss of flesh and strength. If the change from pleurisy with effusion to empyema takes place suddenly, it is usually marked by one or more rigors, followed by a febrile movement.

Physical Signs.—Although the rational symptoms differ in these cases, the physical signs remain the same. These are identical with the physical signs of pleurisy with effusion. There is in the same way, below the level of the fluid, an absence of vocal fremitus, voice, and breathing, and dulness on percussion. At the level of the fluid there is dulness on percussion, and occasionally egophony. Above the level of the fluid there is either normal, exaggerated, or bronchial breathing and voice.

Prognosis.—The termination of this disease is, as a rule, in the death of the patient, or a very slow and incomplete recovery. Of the patients in whom the disease proves fatal, some die in its early stages while the acute rational symptoms still exist. They die at the end of one or two weeks. In others, death takes place from gangrene produced by the decomposition of the products of inflammation within the pleural cavity, and this, as I have said, may occur early or late in the disease. A large number of the patients live for months or years. In some of these cases, if they are allowed to take their natural course, the pus in the pleural cavity will find exit either by perforating the lung, the patient coughing it up from the bronchi, or by perforating the chest-wall and escaping through the intercostal spaces; or, finally, by perforating the diaphragm and finding its way into the abdominal cavity.

During the existence of the disease, which, as I have said, may be for months or years, the patients continue to lose flesh and strength, and become anæmic and suffer from an irregular febrile movement. When the pus finds an exit internally, it is possible for the patients to recover, but with a lung which expands only incompletely and with a pleura which is permanently thickened. It can never return to its normal condition. More frequently these patients finally die from chronic pulmonary phthisis as a complication of the disease, or they may die from exhaustion due to the pyæmia.

Treatment.—In the treatment of empyema it is necessary to meet the indications, which are, attention to the general health of the patient, and the removal of the fluid from the pleural cavity. So far as the removal of the fluid is concerned, the use of diuretics and blisters are of no avail. The only way in which the pus can be removed from the pleural cavity, is by some mechanical means. This may consist either in aspiration or effecting a permanent opening in the chest-wall. If aspiration is resorted to, a needle of large size should be used, and it should be borne in mind that it will be necessary to repeat the operation several times, for the pus will reaccumulate. It will be necessary to aspirate once a week, or once every two weeks. Instead of doing this, a permanent opening in the chest may be made. When aspiration is resorted to, the needle is usually introduced from behind, in the seventh or eighth intercostal space, and nearly in a line with the angle of the scapula. If, however, a permanent opening is to be made, it is better to make the incision more to the side of the chest, in a line with the axilla, which will avoid inconvenience to the patient when lying down. Here again the opening should be made in the seventh or eighth intercostal space. In making such a permanent opening, one of several methods may be employed. With a scalpel a free incision may be made into the pleural cavity, this being kept open by thrusting in pledgets of lint. A smaller opening may be made if a rubber tube is to be inserted, in order to furnish an outlet for the pus. Instead of making a single opening, it may be of advantage to make two, one posterior and the other in front. A rubber drainage-tube may be passed through these. The operative procedures to be adopted in any given case vary with the indications and with the particular fancies of the operator.

In addition to making an opening in this way, the question of washing out the pleural cavity always presents itself—whether it is best to wash out the cavity with simple warm water, or a dilute solution of carbolic acid, or with a solution of tannic acid. With regard to this, however, there is a difference of opinion, some physicians washing out the cavity every day or every second day. The objection to this procedure, however, is that in some cases it is dangerous to the life of the patient. There are at present upon record a very considerable number of cases in which the patients died apparently from the washing out of the pleural cavity. Here, again, there are also cases of death which we cannot understand; but there is no escaping from the fact that the simple washing out of the chest, in cases of empyema, may influence the death of the patient. Hence, if this measure is resorted to, it should be done with the knowledge that it is always possible for the patient to die somewhat suddenly afterward.

For the improvement of the general condition of the patient, the ordinary tonics are administered in connection with the use of cod-liver oil, and, if possible, the patient should be sent to a warm climate. Too much should never be expected from the treatment of cases of empyema. After the chest has been opened, the patients, as a rule, get better for a time and continue to live for months, or years ; but they very seldom get entirely well. Sometimes the patients are apparently well for a sufficient length of time to pass from under observation, but usually they die sooner or later from the exhaustion of the disease, or from its being a complication of chronic pulmonary phthisis.

This prognosis, however, is commonly true in the case of adults. Of the children who have empyema, a large number get well. The indications for treatment in children are different from those in adults. In children it is always advisable to aspirate. As a rule, a permanent opening in the chest-wall is more commonly made in the case of adults.

LECTURE XXVI.

HYDROPNEUMOTHORAX.—PLEURISY WITH ADHESIONS.

Hydropneumothorax; Manner of Occurrence, Symptoms, Physical Signs, Prognosis, Treatment.—Pleurisy with Adhesions; Pathology, Symptoms, Course of the Disease, Physical Signs, Prognosis, Treatment.

GENTLEMEN :—We were speaking yesterday of pleurisy with the production of fibrin, serum, and pus, or empyema, as it is commonly called. We will take up the consideration of this subject at the point where we left off.

HYDROPNEUMOTHORAX.—In a certain number of cases of empyema the condition of the patient is somewhat changed by the admission of air into the pleural cavity. When air is thus admitted patients are said to suffer from *hydropneumothorax*—that is, there is both air and fluid in the pleural cavity.

Manner of Occurrence.—This condition may be brought about in several ways. It occurs most commonly in connection with an opening either in the wall of the thorax or in the lung, so that air gains access to the pleural cavity through the opening. Such an opening may take place in one of several ways : First, a patient may have an abscess in the wall of the thorax which opens externally through the skin, and which, after a time, perforates internally into the pleural cavity. When this is the case the inflammation in the pleura will be secondary, and the condition will be, from the first, one of hydropneumothorax. Second, the primary lesion may be in the lung in the form of an abscess, or the patient may suffer from chronic phthisis, and have a cavity in his lung. The abscess or cavity perforating the pleura, the patient will at once suffer from hydropneumothorax. Third, patients may first have a central empyema, there being no air in the pleural cavity; after a time perforation into the lung takes place and air is admitted; or the empyemic abscess perforates through the walls of the thorax; or the wall of the thorax is opened artificially, and air gains access to the pleural cavity.

Symptoms.—The rational symptoms differ according to the way in which the hydropneumothorax is developed. If the patient has first empyema, and this is changed into hydropneumothorax, there is no change in the symptoms of the patient. He will continue to suffer in the same way as before the admission of air into the chest, but, as we will see, this change will make a difference in the physical signs.

If, however, there is an abscess situated externally and it perforates into the pleural cavity, or if there is a cavity in the lung which ruptures into the pleural cavity and hydropneumothorax is at once set up, there will be, as a rule, marked symptoms attending the commencement of the disease. The patient usually begins to suffer immediately from a severe pain in the chest, which is referred to the part where the inflammatory process is going on. This pain begins apparently at the very moment at which the perforation into the pleural cavity takes place. Patients will often tell you that they experience a feeling as if something had broken or ruptured within them. There is commonly a sudden and very marked dyspnœa. The patients sit up in bed and use all of their respiratory muscles vigorously in their effort to get air into the lungs. The prostration is very great. The pulse becomes rapid and feeble. The skin gets cold and is often bathed in perspiration. The expression of the patient's face is anxious and haggard, and, in some cases, the prostration goes on to actual collapse, and the radial pulse can no longer be felt. The collapse may be so marked that the patient will die in a few hours after the perforation has taken place. More frequently, however, these urgent symptoms will gradually pass off and the patient will slowly become more comfortable, and will then suffer from symptoms of chronic empyema.

In those cases of empyema in which the products of inflammation undergo gangrene, there is still a third variety of hydropneumothorax. This differs from the condition of which we have been speaking, in that the air in the pleural cavity is not atmospheric, but consists of gases produced by the process of putrefaction. The patients in these cases suffer from the presence of both gas and fluid in the pleural cavity.

Physical Signs.—The physical signs of hydropneumothorax differ somewhat from the physical signs of empyema or of pleurisy with effusion. The affected side of the chest is, as a rule, larger than the opposite side. Of course this physical sign is often found in empyema and pleurisy with effusion, but it is more marked and constant in these cases. The affected side usually moves less than the opposite side. This is especially noticeable when the patient attempts to take a full inspiration. This symptom occurs occasionally in pleurisy with effusion and in empyema, but in hydropneumothorax it is almost constant and much more marked.

The percussion-note over the affected side of the chest varies a good deal in different cases, the variation apparently being due to the thickening of the pleura and the amount of fluid in the pleural cavity in proportion to the amount of air. In some cases, when there is a large amount of air and a small amount of fluid in the pleural cavity, there will be tympanitic resonance over the whole of the affected side of the chest, even below the level of the fluid. In other cases the tympanitic resonance will be confined to that part of the chest where there is no fluid. If, for example, the pleural cavity is half-filled with fluid, there will be tympanitic resonance in front and behind, above the level of the fluid. Below this there will be flatness. Frequently, by changing the position of the patient there will be a change in the line of flatness, which is much more marked in hydropneumothorax than in pleurisy with effusion or empyema, and is apparently due to the presence of air in the cavity of the pleura. If you place your ear to the chest, you can usually hear what is called amphoric breathing, which has a peculiar musical sound, resembling that obtained by the blowing of air into the open mouth of a bottle. It is a peculiar, low-pitched, musical sound. Besides this, there may or may not be a change in the voice. The voice may have an ordinary, high-pitched, bronchial character; it may be a low-pitched bronchial voice, or it may be of a peculiar quality, resembling that of amphoric breathing. Both amphoric voice and breathing are heard, as a rule, above the level of the fluid; usually nothing is heard below the level of the fluid. In some cases a peculiar noise is heard. It is a metallic tinkle, as if drops were falling from a height into a vessel of water. In addition to this, if the patient be shaken well and you apply your ear to the chest, you will hear a sound produced by the splashing of the fluid. This physical sign is known under the name of *succussion.* In order to hear this sound to advantage, you should get another person to suddenly shake the patient while you apply your ear to the affected side of the chest. This physical sign, you should remember, is only obtained in hydropneumothorax.

Prognosis.—The prognosis in this disease is, as a rule, bad. When the condition is secondary to empyema, the prognosis is the same as that of empyema, the patient being made no better or worse by the admission of air into the pleural cavity. If, however, hydropneumothorax is secondary to conditions in the lung, or to those in the walls of the thorax, some of the patients die within a few hours after the perforation; others recover from the immediate effects of this and afterward suffer from the symptoms of chronic empyema. They have the same febrile movement and gradual loss of flesh and strength, and may die, as do patients of empyema, from exhaustion or from phthisis, which existed before the development of empyema, or they may develop secondary phthisis.

Treatment.—The treatment is the same as that for empyema, namely: to make an opening into the wall of the chest through which the fluid in the pleural cavity may escape. In those cases, however, in which there are the symptoms of collapse due to sudden perforation, the indications are to administer stimulants in large doses. Patients who die in a few hours after perforation seem to die from failure of the heart's action. The best stimulant is alcohol. We use either whiskey or alcohol by the mouth or hypodermically, determining the dose merely by the force of the pulse.

PLEURISY WITH ADHESIONS.—The last form of pleurisy of which we have to speak is that known under the name of pleurisy with adhesions. It is not very commonly described in text-books, and yet it is a form of pleurisy which most of you will probably have occasion to see in the course of your practice. The lesion may, in some cases, prove fatal. It may begin either as an idiopathic lesion or may follow a previous attack of dry pleurisy, or pleurisy with effusion. The larger number of cases occur in patients who have recovered from pleurisy with effusion, or dry pleurisy.

Pathology.—No matter how it occurs, the lesion is the same and the patients suffer from a chronic inflammation of the pleura. This does not lead to the generation of the ordinary products of inflammation, but results in the formation of new connective tissue which is spread out over the whole surface of the pleura, the larger part of it being arranged so as to form adhesions between the costal and pulmonary pleuras. The lesion is a progressive one. As time elapses there is a larger and larger number of adhesions formed, covering a greater area of the surface of the pleura, and may, in extreme cases, bind the whole of the external surface of both lungs to the chest-wall, so that the spaces between the pulmonary pleura and the walls of the thorax, between the pulmonary pleura and the diaphragm, between the pulmonary pleura and the pericardium, between the pulmonary pleura and the mediastinal, and the pulmonary pleura and the lobes of the lung, will be obliterated by new connective tissue. The lungs can no longer move freely within the pleural cavity, so that their expansion and retraction is very much interfered with.

Symptoms.—The rational symptoms of this condition are the existence of a certain amount of dull pain over the affected part of the chest, which is, however, usually not very severe, being sometimes present and sometimes absent. The patients may suffer from dyspnœa, especially upon exertion, such as going upstairs rapidly, or trying to run. In bad cases, very slight exertion will bring on an attack of dyspnœa, the patients being only able to walk a short distance before it comes on. There may be also a slight, dry cough, without any expectoration, and yet it may be very harassing. If the patients develop bronchitis as a secondary lesion, the cough will be attended with the expectoration of mucus and blood. The patients lose flesh and strength, and in all cases become very much emaciated, as do patients suffering from chronic pulmonary phthisis.

In some cases the heart becomes displaced backward. I do not think it is easy to understand the exact mechanism of this displacement, but, as a matter of fact, it is found from time to time in these cases. The displacement is not usually very considerable; but the apex, instead of being in the fifth intercostal space, midway between the nipple and sternum, will be drawn to the right, approaching the middle line upon the same level, and, in some cases, being fairly opposite the middle of the sternum. When the heart is thus drawn out of its place, the dyspnœa is increased, and in addition the heart is apt to become atrophied and its cavities smaller than normal. This decrease in size is not accompanied by any perceptible change in the muscular fibre of the heart. There is not usually any fatty change in its muscular fibres.

Course of the Disease.—The progress of the disease is, as a rule, slow, and is very often intermittent, there being periods of weeks or months during which patients get better, and gain in flesh and strength. Their appetite increases and there is less dyspnœa, less pain; but, after a time, the symptoms of the disease will return and the patient's health depreciate.

Physical Signs.—There is dulness on percussion, which may not be very marked, but is usually present. If you listen to the patient's breathing, you will find that it is not as loud as normal. Sometimes you will scarcely be able to hear any breathing whatever, even during a forcible inspiration. In some cases you will hear friction-sounds, but these are not like the ordinary ones of acute pleurisy. The sound is a peculiar, loud, creaking one, as if the adhesions were being stretched; often it is like a subcrepitant râle, or mucous râle, or a gurgling one. These sounds are not produced in the lung, but simply by the movement of the pleuritic adhesions and the thickened pleura.

Prognosis.—The prognosis in these cases varies with the extent to which the disease has progressed at the time the patient comes under treatment. If it is not very far advanced, if the adhesions are not very extensive, it is always possible for the inflammatory process to be permanently arrested and for the patient to recover. If the lesion is of very considerable extent when the patient comes under observation, it is more apt to increase. If it does increase, it eventually leads to the death of the patient. A certain number die without the development of any other lesion. They die emaciated and worn out simply by the disease. In other cases the disease becomes complicated by the development of chronic bronchitis, interstitial pneumonia, emphysema, or chronic pulmonary phthisis. In such cases the death of the patient is due only partly to the primary lesion.

Treatment.—The indications for treatment are the use of counter-irritation over the affected side of the chest and the improvement of the nutrition of the patients. They are treated very much as though suffering from chronic pulmonary phthisis. Iron, quinine, and the mineral acids are given to improve the appetite, and cod-liver oil or cream to improve the nutrition. It is well to send them to a warm climate in winter, and to have them travel in summer for the improvement of the general health. During all this time counter-irritation should be employed. The best means of doing this is by the use of iodine painted over the affected side of the chest.

LECTURE XXVII.

STENOSIS AND DILATATION OF THE ŒSOPHAGUS.

Stenosis of the Œsophagus; Congenital, Due to Compression, Introduction of Foreign Bodies, Polypoid Tumors, Cicatritial, Carcinomatous, Spasmodic.—Fusiform Dilatation of Œsophagus; First and Second Varieties.—Sacculated Dilatation of the Œsophagus; Etiology, Symptoms, Treatment.

GENTLEMEN:—To-day we will consider two conditions of the œsophagus, namely stenosis and dilatation.

STENOSIS OF THE ŒSOPHAGUS.—Of stenosis of the œsophagus there are seven varieties. In the first place, there is congenital stenosis, which is a congenital malformation of the œsophagus. It is of rare occurrence, most cases being seen in children who die soon after birth; some, however, live until adult life. When this happens the predominant symptom is constant difficulty in swallowing. This difficulty slowly increases from year to year, and the patients finally die from starvation.

In the second place, there is stenosis due to compression. The ordinary causes of such compression are aneurism of the arch of the aorta, tumors of the thyroid gland, enlargement of the glands of the neck and mediastinum, and intrathoracic tumors. The only symptom is difficulty in swallowing.

In the third place, there is stenosis of the œsophagus due to the introduction of foreign bodies into it, and the impaction of the same therein.

Fourth, there is stenosis due to polypoid tumors growing in the lower part of the pharynx and projecting downward into the upper part of the œsophagus, which they close more or less completely, according to their size. The only symptom is difficulty in swallowing, and the treatment consists in the removal of the tumors.

In the fifth place, there is cicatritial stenosis. This is due to the patient's swallowing irritant substances. The substances most commonly swallowed are the strong acids and the strong alkalies. These set up an inflammation more or less destructive to the walls of the œsophagus. To replace the destroyed tissue, there is a production granular tissue and afterward of connective tissue. By the contractio of the cicatrices a stenosis is produced, sometimes over a small pa of the length of the œsophagus, and at other times over a very larg part of its length. The stenosis may be very great, or only moderat

The symptoms are: first, the acute symptoms consequent upon th inflammation produced by the poison, and, after these have subsided, the inability to swallow anything but fluid food. The degree of th dysphagia varies with the extent of the stenosis. The treatment consists in the gradual dilatation of the stenosis by means of œsophageal bougies. The treatment is very difficult and tedious, requiring a great deal of manual skill, and is not always successful.

In the sixth place, there is stenosis of the œsophagus due to syphilitic inflammation. This is a very rare condition.

In the seventh place, there is stenosis due to carcinoma. This is the most common form.

Pathology.—The new-growth resembles in its anatomy the epithelial carcinomata of the skin. It usually surrounds the œsophagus and involves one or two inches of its length; occasionally it involves the whole length of the œsophagus. There is regularly a certain amount of ulceration, and this may be out of proportion to the new-growth. In most of the cases the new-growth is situated in the middle and lower third of the œsophagus. It may extend to and infiltrate the surrounding tissues, especially the trachea, bronchi, lungs, pleura, and pericardium. There may be metastatic tumors in the lymphatic glands, lungs and liver. The same patient may suffer from cancer of the œsophagus and cancer of the stomach. The disease is much more frequent in males than in females, and it rarely occurs before the age of forty.

The symptom dysphagia comes first, and it may exist for months without any other symptom. It is progressive. The patient manifests more and more difficulty in swallowing, and as time elapses there may be a regurgitation of food from the œsophagus into the mouth. When ulceration supervenes the dysphagia may improve for a time, and then come on again. The pain, as a rule, is referred to the upper end of the sternum. Sometimes it begins early in the disease, at other times, not until late, and still again there may be no pain at any time.

The patients usually die within a year, and most of them from starvation. Some, however, may die from infiltration of the new-growth into the neighboring tissue.

Lastly, there is spasmodic stricture of the œsophagus. Here there is no lesion, the stricture being due to the contraction of the muscular coat of the œsophagus. It occurs more frequently in women than in men, and usually during adult life. The dysphagia is only partial, or it may be almost complete. It comes on suddenly, and is well-marked at once. It may last for only a few minutes, or it may last for hours, days, or months. The same person may have a number of attacks.

The immediate treatment consists in the passage of the œsophageal sound. To prevent the reoccurrence of the attacks, the general health of the patient should be attended to.

DILATATION OF THE ŒSOPHAGUS.—We come now to the dilatations of the œsophagus, and of these there are two kinds:

Fusiform Dilatations.—The first variety is the fusiform dilatation. Sometimes this is secondary to stenosis of the œsophagus, and sometimes it is idiopathic. It may involve only a small part of the length of the œsophagus, or its entire length. The wall of the œsophagus is usually thickened.

The symptoms of this condition are difficulty in swallowing, vomiting, regurgitation of food, and rumination. The treatment consists in feeding the patient by means of the œsophageal tube.

Sacculated Dilatations.—The second variety of dilatation of the œsophagus is the sacculate or diverticulate. There are two kinds. The first are situated at the juncture of the pharynx and the œsophagus, on the posterior wall of the œsophagus. They are at first very small, but gradually increase in size, forming large sacs which hang down behind the posterior wall of the œsophagus and compress it, so that whatever is swallowed passes into the sac rather than through the œsophagus.

Etiology.—As to the causation of this disease, it is supposed by some that it is a congenital condition. By others it is thought to be due to some mechanical cause which destroys the power of resistance of some part of the posterior wall of the pharynx. It is more commonly seen in men than in women, and, as a rule, occurs late in life.

Symptoms.—The first symptom is difficulty in swallowing. As the sac becomes larger and more and more food passes into it, and less and less into the stomach, the œsophagus is compressed to a greater degree by it. After the food has remained in the sac for some time, it is regurgitated into the mouth, and the patients chew it over again. If the sac is large enough, it can be felt as a tumor in the neck, and when you try to pass the œsophageal bougie there is always great difficulty in getting it into the œsophagus instead of into the sac.

Treatment.—The treatment is to feed the patients by means of the stomach-tube, but the cases all prove fatal after a certain length of time.

In the second form, the diverticuli are small and are situated upon the anterior wall of the œsophagus, at its middle part. They are due to a preceding inflammation of the bronchial glands. The bronchial glands become inflamed and adhere to some part of the anterior wall of the œsophagus, and they draw this part of the wall of the œsophagus downward, and in this way a small sac is formed. These diverticuli do not give rise to any difficulty in swallowing, and they cannot usually be made out during the life of the patient; but they sometimes cause death by perforation and allowing the food to escape from the œsophagus into the surrounding tissue, thus setting up an inflammation which proves fatal.

LECTURE XXVIII.

LESIONS OF THE STOMACH—GASTRIC DYSPEPSIA.

Position of the Stomach; Anatomy, Function.—Kind of Food Digested by the Stomach.—Gastric Dyspepsia; Etiology.

GENTLEMEN:—We come now to the consideration of the lesions of the stomach.

Position of the Stomach.—The stomach is situated, as you know, in the upper part of the abdominal cavity. When empty, it is overlapped by the left lobe of the liver and lies in front of the pancreas. The points about the situation of the stomach which it is necessary to remember, are the position of its œsophageal and pyloric extremities, its greater curvature, and, to a certain extent, its lesser curvature. The natural position of the œsophageal or cardiac end of the stomach is a little to the left of the sternum, and about on a level with the cartilage of the sixth rib. No matter what disease the stomach may be the seat of, this is its most fixed point. The pyloric end is about on a level with the seventh intercostal space, and lies underneath the free border of the ribs, being naturally covered by the left lobe of the liver. In a healthy person it ought not to be possible for the pyloric end of the stomach to be felt. This part is not very fixed. It is very apt, in diseases of the stomach, to become dislocated, being most frequently displaced downward. The pylorus may be found low in the epigastrium, and it has been found as low as the iliac region, either in the median line or to its right. The position of the greater curvature of the stomach, of course, varies with the amount of food which the stomach contains. Ordinarily it comes down below the free edge of the ribs, not quite to the umbilicus. This is when it is moderately distended. If the stomach contains more food, it may come down on a level with the umbilicus, and this without any abnormal condition. If, however, the stomach is habitually as large as this, it constitutes an abnormal condition.

Anatomy.—When we come to the structure of the walls of the stomach, we have to remember that there is on the outside a serous coat derived from the peritoneum; beneath this there is a muscular coat; next in order there is a loose connective-tissue coat (cellular), and finally, upon the inside, there is the mucous coat and in it a number of tubular glands are situated which perform the function of the stomach.

Function.—Although they have the same general shape, these glands have two distinct functions. A certain number produce simply mucus; another set, which comprises the larger number, produce only gastric juice. This difference in function gives to inflammations of the stomach certain characteristics. The parts which are principally involved in morbid processes are the mucous and connective-tissue coats. The gastric tubules are not situated close to each other, but they are separated by connective tissue and capillary blood-vessels. It is this connective tissue between them which is apt to undergo inflammatory changes. The regular function of a healthy stomach is to take a share in the digestion of food. You know that digestion is carried on by means of the stomach, intestines, liver, and pancreas. The particular share which the stomach has in the digestion of food is the conversion of a certain part of it into a material which will pass into the intestines and will be absorbed by them. In performing this function, there are two anatomical parts of the stomach which are concerned; in the first place the glandular coat, and in the second the muscular coat. The muscular coat has the function of moving the food about in the stomach, and finally expelling it. The gastric tubules have the function of liberating the gastric juice, which acts upon certain elements of the food and converts them into a material that can be absorbed by the small intestine. The particular parts of the food which are acted upon by the gastric juice are what are called the nitrogenous or albuminous parts.

There is found in ordinary articles of food a certain amount of nitrogenous substance which must be digested by the stomach, if it is to be of any use to the patient. In ordinary flour, the particular part which is digested is the gluten; in meat it is the fibrin. The same is the case with fish. It is the fibrin of the muscular tissue of the fish that is acted upon by the stomach. In eggs there is nothing to be digested except albumen. In milk the particular part which is acted upon by the stomach is casein. So that, of the ordinary articles of diet, there are certain parts to be digested by the stomach and certain other parts which are not to be so digested.

GASTRIC DYSPEPSIA.—In ordinary practice, we meet very frequently with cases of what are called dyspepsia, or indigestion. Of these, we can distinguish cases which are due to conditions, not of the liver or intestine, but of the stomach; and these we call, for convenience, by the name of *gastric dyspepsia*.

Etiology.—The causes of gastric dyspepsia are, in the first place, the ingestion of improper food. Dyspepsia due to this cause is usually developed pretty slowly. It occurs in persons who, for a considerable length of time have been in the habit of taking food which the stomach could not digest; sometimes it is due to the character of the food, and at other times to the ingestion of excessive quantities. In either case the effect is the same. There is introduced into the stomach a mass which is not acted upon by the gastric juice, or only imperfectly acted upon, at best. This food, remaining undigested in the stomach, does harm in two ways. In the first place, it acts as a foreign body, directly irritating the mucous membrane, and in this way the function of the mucous glands of the stomach is unduly excited and there is an increased production of mucus which coats the inner surface of the stomach and prevents the gastric juice from coming in contact with the food as it should. This increased amount of mucus is liable to decompose, somewhat in the same way as mucus in the bladder. Besides acting as a foreign body and an irritant, the food itself may become decomposed to a certain extent. It may ferment, with the evolution of gas, and this adds to the irritation of the mucous membrane. Another cause of this form of dyspepsia is the production of gastric juice of an abnormal composition, which is incapable of digesting the food properly, or of an abnormal quantity. A person may eat perfectly proper food and in proper quantities, and yet, if the stomach does not liberate gastric juice in such quantity and of such quality as to digest the food, he will suffer from dyspepsia. Such an abnormal condition of the gastric juice may be due to nervous influences—to mental conditions. Persons whose health is bad from any cause may also have gastric dyspepsia, the stomach suffering the general malnutrition of the body, and the gastric juice not being secreted in proper quantity or of a proper quality. Another cause is structural disease of the mucous membrane. It is found that inflammations affecting the mucous membrane of the stomach may be accompanied by degenerative changes leading to atrophy of the gastric tubules, and there may be ulcers and new-growths in the walls of the stomach. These will lead to gastric dyspepsia. There is still another cause to be found in the abnormal condition of the muscular coat of the stomach. If this becomes weak, flabby and relaxed, the food cannot be properly moved about, and the stomach cannot be properly emptied. If the muscular coat becomes thickened at the pyloric end of the stomach it will lead to difficulty in the escape of food, and if there is a general thickening of the muscular coat of the stomach it will interfere with the function of the affected parts. These are the ordinary causes of gastric dyspepsia.

LECTURE XXIX.

LESIONS OF THE STOMACH—CONTINUED.—GASTRIC DYSPEPSIA.

Gastric Dyspepsia; Local and General Symptoms, Treatment.

GASTRIC DYSPEPSIA.—*Gentlemen:* We will continue to-day the study of gastric dyspepsia, and consider first, the symptoms. These are of two kinds, local and general.

Local Symptoms.—Of the local symptoms it is noticed, in the first place, that there is a change in the appetite in one of three ways—it is diminished, increased, or perverted. In many cases of gastric dyspepsia the appetite is simply diminished, the patient not caring to take a proper quantity of food, and if left to himself will hardly take any food whatever. Even the sight of food excites in some patients a certain loathing, and it is only by considerable effort of will that they are enabled to take sufficient food to support life. In other patients exactly the opposite condition is found. They want to eat nearly all the time and take food in large quantities. They find that they are more comfortable when the stomach is full than when it is empty. In still other cases the appetite is perverted, the patient craving improper food, sometimes acids, at other times, especially in young women, articles like paper, slate-pencils, and chalk. Besides a change in the appetite, there may be pain, which will vary considerably in character and constancy. Sometimes there is a feeling of oppression rather than of real pain. Patients feel as if there was a load in the region of the stomach, or as if there was something pressing upon the stomach. Others experience a peculiar feeling of emptiness in the region of the stomach. Again there may be a dull, aching pain, or a pain that may be compared to a dull toothache. In other cases it is described as a gnawing pain, or of a sharp lanceolating character. In some instances, the pain is almost continuous. The patients will tell you that it never leaves them, although it is worse at some periods than at others. The pain may be intermittent, and it usually has some relation to the ingestion of food. In some the pain is more severe when the stomach is full and is relieved when it is empty. In other cases the ingestion of food relieves the pain.

Sometimes the pain is referred to the epigastric region, sometimes to the left hypochondrium or stomach, and sometimes to the left shoulder-blade, and over the region of the sternum. The amount of pain is no very great criterion as to the amount of stomach disease. It seems to depend partly upon the stomach disease and partly upon the individual himself. There are some persons who always manage to experience a great deal more pain than others from any lesion they may have. The symptom of pain is a bad one so far as treatment is concerned, but is no indication of the extent of stomach disease. Another common symptom is nausea and vomiting. Nausea is especially common. Patients feel a little sick at the stomach without actually vomiting. This is much more common than actual vomiting of food. The time when nausea is most frequently experienced is early in the morning and it is often accompanied by considerable mental depression. When the patient wakes in the morning the first thing he feels is a sickness at the stomach and it has a depressing influence upon the spirits. If this happens every morning, the mental condition of the patient is a good deal affected by it. Vomiting is less common and usually indicates a worse form of dyspepsia. Its immediate cause seems to be, in some instances, the mere presence of undigested or fermenting food in the stomach. In other cases, however, the cause of vomiting does not seem to be within the stomach but is to be referred to the condition of the nervous centres which bring on the act of vomiting. The matters vomited are sometimes formed of food. In other cases there will be an increased amount of mucus produced in the stomach or there may be present a considerable amount of thin, brownish fluid which is derived from the mucous membrane of the stomach. In some cases there may be vomiting of blood. If it is customary for the food to ferment, there may be found in the vomited matter microscopic organisms and sometimes the so-called sarcina ventriculi, and the various forms of bacteria. There is one form of vomiting called *water brash*, which usually takes place in the morning, and is easily effected, the patients having to make very little effort. It consists of a peculiar thin, sour, fluid which is regurgitated. Another symptom of gastric dyspepsia is flatulence, a distention of the stomach and perhaps also of the intestine with gas, which seems to be produced by the fermentation of the food. The gases which are usually found are carbonic acid gas, carbonated and sulphurated hydrogen gas and hydrogen gas. This symptom usually troubles patients in two ways; in the first place, by causing a feeling of oppression which is very distressing. The distended stomach presses upward upon the thoracic viscera, the oppression being referred to the heart or lungs, and gives rise to much anxiety on the part of the patient. Another annoying symptom is flatulence with constant belching of wind from the stomach into the mouth. In some cases this is so offensive that these patients are obliged to keep away from other people as much as possible.

General Symptoms.—There are in a certain number of cases cerebral symptoms, and of these the most common perhaps is headache. There are a great many varieties of headache due to gastric dyspepsia. Sometimes it is a dull pain over the whole of the head, or it may be circumscribed. Sometimes it is a neuralgic pain. The headache may be constant or intermittent. It may come on every day or every few days, or continue for a week. More frequently, however, the headache only comes on at a certain time. Besides this pain in the head, the patients may have abnormal sensations in the head. At times, the top of the head feels as if it had a piece of ice upon it. Sometimes the patients will tell you that a part of the head feels hotter than it should. Again, they complain of a loss of memory, or they may complain of dizziness, or of vertigo. Some patients have attacks of vertigo from time to time, and in its worst form it may terminate in complete unconsciousness. Patients become so dizzy that they will fall to the ground and remain unconscious from a few moments to half an hour, the loss of consciousness being due simply to the condition of the stomach. There are also symptoms referable to the thorax. Occasionally, there is not only a feeling of oppression in the thorax, but there is actual pain, which is referred to the heart or lungs. The functions of these organs may be interfered with, the patients suffering not infrequently from palpitation of the heart and dyspnœa. When this is the case, they are very apt to think they are suffering from heart disease, or when shortness of breath is present they think they are suffering from some disease of the lungs. There are also symptoms referable to the abdominal viscera, pain being referred to the lower part of the abdomen instead of to the stomach. In women, it is not infrequently referred to the uterus, and leads them to think that they are suffering from uterine disease, and they are sometimes treated for this when the real trouble is gastric dyspepsia. The bowels are very apt indeed to be constipated, there being a sympathy between the functions of the stomach and the large intestines. This sympathy is manifested in two ways. In some patients with gastric dyspepsia, as I have said, there is constipation; others have a sort of diarrhœa, not due to the condition of the colon, but to that of the stomach. The converse of this proposition sometimes obtains in diseases of the colon. In addition, there are changes in the general health of the patients. They may lose flesh and strength and become anæmic, simply as a result of prolonged interference with the functions of the stomach.

Treatment.—The treatment of gastric dyspepsia is partly rational and partly empirical. We should try to determine the cause of the dyspepsia and find out if possible whether it is due to improper food, to an abnormal condition of the gastric juice, to actual disease of the mucous membrane of the stomach, or to changes in the muscular wall of the stomach. If the cause can be clearly made out, the treatment will be much easier and more satisfactory; but such definite information cannot always be obtained.

If the dyspepsia is dependent upon improper food, the only thing necessary is the regulation of the patient's diet, as to the kind of food, its quantity, and the time at which it is taken. If the case has lasted for a very considerable length of time, it is commonly necessary to put the patient upon a milk diet, and give the stomach, as nearly as possible, complete rest. If, however, it is not of long duration, it may be sufficient to simply restrict the patient to ordinary food. He should be allowed to take meat, plain bread and butter, and a certain amount of vegetables and fruits. Such patients are to eat only a moderate amount of food at each meal, which should consume a fair amount of time. In this way they can usually be relieved of the symptoms in a moderate length of time. If the dyspepsia is due to an abnormal condition of the gastric juice, and this is dependent upon the general condition of the patient and not upon structural disease of the mucous membrane, the treatment should be directed to the improvement of the patient's general health rather than to the stomach. In these cases the use of regulated exercise is very beneficial. If the patients are not well enough to take active exercise, the muscles may be rubbed and electricity applied to them until they are sufficiently strong to take exercise. It is best to exercise out of doors. In-door exercise is of comparatively little service in the treatment of these cases. Such patients are always benefited by travel. The climate is of no particular consequence, but simply moving about from place to place is usually of great benefit. Of the tonics, the bitter tonics, strychnine, nux vomica, iron, quinine, and the mineral acids are all of service. The use of alkalies before eating will very frequently apparently stimulate the production of gastric juice. Bicarbonate of soda or potash, given in some bitter infusion before meals, will, in a certain number of cases, aid digestion, and apparently the production of gastric juice. The patients will only be able to take at each meal a small amount of food to be digested by the stomach ; in other words, they should be fed upon fats and starches. In other cases this form of feeding will not answer at all. Instead of fats and starches it will be found that patients do much better upon a small amount of easily digested food. It is very difficult to say what plan of treatment will produce the most favorable result. There is no general rule, the plan of feeding being determined only by trial.

If gastric dyspepsia is due to disease of the mucous membrane, the most common lesion is that of catarrhal inflammation of this structure. Here, again, it is necessary to regulate the food and to determine by trial whether the patient does better upon principally stomach food or principally intestinal food. Of the drugs which are usually given for the relief of such inflammation of the stomach the most common are the preparations of bismuth, nitrate of silver in small doses, preparations of belladonna in small doses, the mineral acids, and alkalies. Sometimes acids are of more service, sometimes alkalies. If the gastric dyspepsia is due to the condition of the muscular coat of the stomach, especially if it is relaxed, patients do best upon a strict milk diet for a time. If this does not afford relief it will be necessary to empty the stomach every day, or every second day, so as to allow the relaxed muscles to contract, for there is, under such circumstances, a tendency for the food to accumulate.

LECTURE XXX.

LESIONS OF THE STOMACH—CONTINUED.—ACUTE AND CHRONIC CATARRHAL GASTRITIS.

Acute Catarrhal Gastritis; Etiology, Symptoms, Prognosis, Treatment.—Chronic Catarrhal Gastritis; Pathology, Etiology, Symptoms, Treatment.—A Peculiar Form of Chronic Catarrhal Gastritis; Physical Signs, Rational Symptoms, Treatment.

ACUTE CATARRHAL GASTRITIS.—*Gentlemen:* We now come to inflammations of the stomach, and we will consider, first, acute catarrhal inflammation of the stomach—*acute catarrhal gastritis*. In this we have the same changes in the mucous membrane of the stomach that we have in any mucous membrane when it becomes the seat of catarrhal inflammation. It looks redder than normal and there is a certain amount of swelling and an increased production of mucus, so that the free surface of the membrane is coated with it. There is also a moderate production of pus-cells in the stroma of the mucous membrane. These lesions, not being structural, may not persist after death. The most persistent of them, however, is the mucus which is usually found at the autopsy. In a normal stomach after death, there is found only a little mucus coating the pyloric end, but in patients who die of acute catarrhal gastritis, the stomach may be found extensively coated with mucus, not only over the pyloric end, but over its whole surface.

Etiology.—Acute catarrhal gastritis occurs at all ages and in both sexes. It is more common in summer than in winter. Persons who suffer from fatty infiltration of the liver are more apt to have it than others. In the disease ordinarily known by the name of cholera morbus, a part of the lesion consists in an acute catarrhal inflammation of the mucous membrane of the stomach, but in this affection some parts of the small and large intestines are usually also involved.

Symptoms.—The symptoms of this condition are pain, vomiting, a febrile movement and prostration. There is considerable difference as to the predominance of one or other of these symptoms, according to the age of the patient. In *infants*, the most prominent symptom is apt to be the febrile movement. If there is any pain at all, these children are so young that they are not able to make its existence known, and most commonly they do not seem to suffer. Vomiting is generally present and may be very frequent. There is not very much prostration if the child is moderately healthy at the time of the attack. The thermometer may indicate a temperature of 103°, 104°, or 105°, and if you were guided alone by this you would be very apt to think the patients were suffering from some grave disease, whereas it is only an inflammation of the stomach, which is, as a rule, of short duration. In *children* the most prominent symptom is pain. There may be a little febrile movement or none at all. There is not much prostration, but the particular symptom of which the child complains is pain referred to the epigastric region and upper part of the abdomen : not usually to the region of the stomach. In *adults* the most marked symptoms in the majority of cases is prostration. There is no febrile movement whatever. There is vomiting, and with it very frequently pretty marked prostration, the patients feeling so weak that they go to bed. The pulse becomes very feeble, the skin is cold, the expression of the face anxious, and eventually the patients are very much prostrated by the inflammatory process.

In patients who are already suffering from fatty liver there is commonly, in addition to the gastritis, a duodenitis, the patients going on to suffer from gastro-duodenitis, a condition of which we will speak at a later period.

The duration of the inflammatory process is not very long, being from twenty-four hours to ten days, most cases running their course within a week.

Prognosis.—The prognosis, as a general thing, is good. Infants and children who have previously been badly fed, and whose general health is bad, may sometimes die simply from acute catarrhal gastritis ; but if they have been in good health, and are well nourished, there is no danger in this disease. In adults, the only danger is in those cases where there is already a fatty liver. If such patients acquire acute gastritis, they become very ill, and may die from the effects of the disease.

Treatment.—The treatment of this disease varies again with the age of the patients. In infants, although there is a marked febrile movement, it does not require treatment. It is not necessary to give quinine, or a cold bath, or to use any active measure to reduce the temperature, for it does not last long, and does not seem to do much harm. For the vomiting it is well to give either an alkali or a small dose of calomel. The bicarbonate of soda or potash may be given in milk. If the children are old enough to be fed artificially, or have been brought up on a bottle, I think the best way to administer the alkali is in cream mixed with water. One part of cream with two, three, or four parts of water, to which is added, two or three, or four grains of bicarbonate of soda, according to the age of the child, may be given every three or four hours. In a certain number of cases, this will be all the medication required. The vomiting will gradually subside, the febrile movement disappear, and the child return to a natural condition. In other cases, patients do better upon small doses of calomel. The tenth or twelfth of a grain of this should be placed upon the tongue with a little sugar, and given every three or four hours. These two drugs, I think, are of greater service in the treatment of the disease in infants than any others.

The treatment of children is somewhat different. There is not, usually, very much febrile movement, but there is a certain amount of pain and vomiting. The vomiting, as a rule, is best controlled by the administration of a mixture of equal parts of milk and cream in a tumbler, to which is added ten grains of bicarbonate of soda, the patient taking no food except this. For the relief of pain, small doses of opium should be given in the form of Dover's powder, giving just enough to keep the child tolerably comfortable while the inflammatory process exists. If the bowels are constipated, they may be relieved by the use of enemata or mild cathartics, such as mineral waters. It is not necessary to purge these children to any extent.

Adults are fed either exclusively upon milk, or a mixture of milk and cream as in the case of children, and as with children there is added either bicarbonate of soda or potash. In addition to this, adults may often eat with advantage dry toast, arrow-root, or other starchy food. Soups and broths are usually not very well borne by them. Ordinary beef-tea and chicken-broth, as a rule, do not agree with these patients. Chewing and swallowing of small pieces of ice is often very agreeable to the patient, and has a certain effect in checking vomiting. The prostration from which the patients suffer often indicates the use of stimulants. These should be administered diluted. Generally speaking, one of an effervescent nature is best, such as dry champagne, which will aid in checking the vomiting, and at the same time furnish the necessary stimulation.

Infants, children, and adults suffering from this condition should be kept as quiet as possible, not being allowed to go about until the symptoms have entirely ceased; otherwise the symptoms may be protracted for a much longer time and the patients continue to have a certain amount of nausea and feeling of ill-health. If they are kept quiet they recover completely.

CHRONIC CATARRHAL GASTRITIS.—Besides acute catarrhal gastritis, we also very frequently meet with chronic catarrhal gastritis. In this, as in all other chronic inflammations of the mucous membranes, the gross appearances vary.

Pathology.—The stomach is usually coated with an increased amount of mucus, which in some cases is very marked. In this may be embedded a certain number of pus and epithelial cells. The mucous membrane may be of a pale or whitish color, or of a peculiar dark-grayish slate color. It is usually thicker than it should be, but this is not always a diffuse or uniform thickening. It may be punctate, giving the appearance of numerous little prominences. Sometimes there are little polypoid tumors (hypertrophies of the mucous membrane) projecting into the cavity of the stomach. At other times, instead of being thickened, the mucous membrane is thinner than normal. The glandular tubules may atrophy and the cells which fill them may degenerate and be transformed into granular matter, so that when you examine a section of such a stomach, you will not see as many gastric tubules as you should, and those which you do see will be smaller than normal, and deformed in various ways. There is often a new growth of connective tissue taking place between the gastric tubules, and there may be also an increase of the connective tissue between the mucous and muscular coats of the stomach. Sometimes this new growth of connective tissue is very marked indeed. It may be confined to the pyloric end or extend over the whole of the stomach. The muscular coat is often changed. It may be hypertrophied or atrophied. The hypertrophy may involve only the pyloric end, or less frequently the whole of the stomach. When the muscular coat is atrophied it is usually diffuse, involving the whole of it. In still other cases the muscular coat is neither hypertrophied nor atrophied, but simply soft and relaxed. The size of the stomach is not infrequently changed. Sometimes the whole stomach becomes smaller than natural. In other cases the opposite of this condition is found—it is dilated.

Etiology.—This form of inflammation of the stomach occurs usually in adults or old persons; it is never observed in infants and seldom in children. It seems to be due in some instances to the habitual use of improper food or the habitual use of alcohol. Again, in some cases it appears to be due to the prolonged use of drugs of various kinds. It occurs as a complication of liver disease, and chronic disease of the heart, lungs, and kidneys. It also occurs as a complication of cancer and ulcer of the stomach. When cancer or ulcer of the stomach exist there is apt to be chronic inflammation of the rest of the stomach.

Symptoms.—The symptoms of this disease are those of dyspepsia. They are the same as those which I have enumerated as belonging to gastric dyspepsia. The appetite is changed; there is pain, nausea, vomiting, constipation of the bowels, and gradual loss of flesh and strength.

Treatment.—These cases are treated more for the symptoms than for the disease. There is no means of acting directly upon the disease. The best that can be done is to use all available means for improving the general health of the patient. In this way the symptoms will disappear as the health improves. The means for the treatment of the different symptoms I have spoken of in connection with gastric dyspepsia.

Chronic Catarrhal Gastritis with Dilatations of the Stomach.—There is one form of chronic catarrhal gastritis which deserves especial mention. It is that form which is accompanied with a peculiar irritable condition of the stomach and dilatation of the organ. In these cases there are the ordinary lesions of catarrhal gastritis, but in addition there is dilatation of the stomach, it being constantly larger than normal.

Symptoms.—The evidences of this condition are partly physical signs and partly rational symptoms. The plainness of the physical signs will depend very much upon the degree to which dilatation of the stomach has taken place. The most reliable sign of such dilatation is obtained by palpation. If the stomach is dilated it will reach down into the abdominal cavity, and if, as is usually the case when dilated, it contains a certain amount of fluid and food, by placing the hands upon the abdomen in the epigastric region and to the left side, and pressing backward and forward, a peculiar splashing sound will be heard, due to the movement of the fluid and food in the stomach. In determining this condition a certain amount of caution is necessary. If a patient applies for treatment in whom you suspect dilatation of the stomach, and if, upon examination, you do not get this sign, you should not stop here. You should ascertain how long since he has vomited, for if the stomach is empty, or nearly so, this may be sufficient cause for not obtaining the sign sought for. You should cause him to drink a considerable amount of water, when, if dilatation of the stomach exists, you will be able to obtain, upon a second examination, diagnostic signs. Besides palpation, percussion may be used, but this is always somewhat unreliable, for the percussion note of the stomach is very much the same as that of the intestine, especially that of the colon. There is also some difficulty in distinguishing by means of palpation between a dilated stomach and a dilated colon. You may get a splashing sound in either case, but in different places; in dilatation of the colon it is obtained beneath the free border of the ribs and upon the right side. It is possible, however, for the colon to be dilated so that this sound may be heard in the epigastric region, but this is exceptional. Some help as regards percussion may be obtained by distention of the stomach with gas. This can be done by the administration of a seidlitz powder, after which the outlines of the stomach can be determined by percussion. In extreme cases the dilatation is so great as to form an abdominal tumor which can be perceived with the eye. In these cases the pylorus is usually dislocated, and the position of the tumor formed by the dilated stomach is changed accordingly. To this condition there is, as a rule, added to the other lesion, an actual stenosis or narrowing of the pyloric orifice, which is a result of the chronic inflammatory process. This forms a mechanical impediment to the escape of food from the stomach into the intestine.

As regards the rational symptoms of this condition of the stomach, there are to be distinguished three sets of cases. In the first place, there are some patients in whom the most marked symptom is pain. This is of a peculiar character, and depends entirely upon the presence of food in the stomach. If there be no food in the stomach there will be no pain whatever. Besides pain, such patients are also troubled with vomiting, though this is less constant and less marked than the pain. The disease usually comes on very gradually, the patients having at first the symptoms of ordinary gastric dyspepsia. They may get better, for a time, under treatment or change in climate, but after a while the symptoms will re-occur, the patients will get worse, and so for several years may continue to suffer more and more from the ordinary symptoms of gastric dyspepsia. After a time, the patients will begin to notice that the most prominent symptom is pain, which comes on after taking food. At such times they may vomit and observe that the pain is diminished thereby until the next meal. The patients, finding this out, are very apt to get into the habit of artificially causing themselves to vomit, for, by so doing, they find relief from pain. They accomplish this by putting their fingers down their throats. Sometimes the vomiting will take place spontaneously. During the continuance of these symptoms, the patients are getting worse, notwithstanding they are usually being treated with the ordinary remedies for gastric dyspepsia. Or, perhaps, they think they are getting well, or are well, when these symptoms will re-occur and continue to get worse. After a time they confine themselves to a milk diet, and this again relieves them temporarily. They may be better for several months, sometimes for a year, and rarely for more than a year. But, after a time, the milk diet also will fail to relieve them, and the pain will return, for the relief of which the patients will cause themselves to vomit. When they have reached this stage they are in a very pitiful condition. They are emaciated, feeble, anæmic, and the pain is so great and continuous that they are hardly able to sleep at all, the pain continuing during the whole of the twenty-four hours, and is no longer relieved by vomiting. Under these circumstances, they are very apt to seek relief in the use of opium in large and increasing doses. In this way a certain number of these patients become victims of the opium-habit. The symptoms constantly progress, continuing, with intermissions, to get worse until the patients suffer from acute pain during nearly the whole of the twenty-four hours, which is not relieved by anything except large doses of opium.

In the second set of cases the symptoms are somewhat different, although there exists the same lesion in the stomach. In these cases, the progress of the disease, as before, is slow, the patients merely complaining, at first, of the ordinary symptoms of gastric dyspepsia. After a time, however, they suffer from a peculiar feeling of oppression in the region of the stomach. The pain is not acute; properly speaking, it is not pain at all. It is simply a feeling of oppression which continues to become more and more marked, and finally, there comes a time when the patients begin to vomit spontaneously, and a great deal. The vomited matter is composed of food, digested or undigested, of fermented food, and sometimes it contains sarcina ventriculi and bacteria. After having vomited once in this way, the patients feel more comfortable for some time, and may not vomit again for a week or two. After a while, a feeling of oppression is experienced and increases until vomiting again takes place, and so these patients continue to vomit at intervals. As the disease progresses, the vomiting occurs at shorter intervals, and in the worst cases, the patients may vomit every second day, or every day. A considerable number of patients continue for a long time to vomit not oftener than once a week. The general health of the patients does not seem to suffer much. They may have a good color, and their muscular strength may continue intact.

In the third set of cases the most marked of the symptoms is loss of nutrition. It is in these cases that there is a real narrowing of the pyloric orifice, this condition not being met with in the preceding cases. The patients may have the symptoms which belong to the first set of cases or those belonging to the second set of cases ; but, added to these, there is rapid emaciation of the patient and persistent constipation of the bowels. There is indeed constipation of the bowels in the first and second set of cases, but it can be relieved by the use of enemata or drugs. In the third set of cases the constipation is apt to be very persistent, and does not yield to the use of drugs or enemata. In a certain number of these patients, in addition to the other symptoms, there will be cramps of some of the voluntary muscles, especially those of the flexors of the arms. There may be also spasmodic contraction of the abdominal muscles.

Treatment.—The treatment in the first set of cases consists in emptying the stomach by means of the stomach-pump or stomach-tube, and after so doing, in washing it out. At first this is to be done every day or every second day, but may be done less frequently as the patient gets better. The object in doing this is to thoroughly and completely empty the stomach, or, in other words, to accomplish what the patient tries to accomplish, though ineffectually, by vomiting. No matter how much they may vomit, they are unable to empty the stomach altogether, and hence small fragments of food remain in the stomach and continue to give rise to pain. The emptying of the stomach may, as I have said, be accomplished by means of the stomach-pump or funnel. The latter is, perhaps, the most convenient, more especially because patients can be taught to use it themselves. For this purpose an ordinary œsophageal sound of small size is necessary. There is some difficulty in introducing this, especially at the lower part of the pharynx, owing to the contraction of the pharyngeal muscles, which seize the tube. It is with difficulty that the tube is introduced into the stomach of irritable and nervous patients. Several trials may be necessary before the tube can be successfully introduced. As soon as the patients become accustomed to its introduction there is no further trouble. There is some little difficulty in New York in getting a stomach-tube of the right kind. The calibre is too small, as a rule, and not only this, but the openings at the end of the tube are also apt to be too small. The best tubes are those which are not stiffened by wire, but which are made of some material which is stiff when it is cold and flexible when placed in warm water. Most of the stomach-tubes in market are too short. They should be long enough, so that when introduced into the stomach one end will project several inches out of the mouth. After it is introduced, a piece of rubber tube about four feet long is attached to the end, and into the end of this is inserted a small funnel, into which the water is poured when the stomach is being filled. When the stomach is to be emptied, the tube is converted into a siphon. At first, the funnel is held at the level of the patient's stomach, gradually raising it until the water flows pretty freely into the stomach. The funnel is now lowered, taking care not to let it get empty. Pour in water until the funnel is below the level of the stomach, when, if allowed to discharge itself, the siphon will empty the stomach completely. This process is continued until the water which flows from the stomach is no longer colored. It is then certain that the stomach is completely empty. When carrying on such a plan of treatment it is necessary to place the patient upon a certain diet, and the best is the following: in the morning, either milk alone or mixed with some sort of porridge, as oatmeal. Three hours after breakfast the stomach is to be washed out as above directed. After washing out the stomach, in the middle of the day, the patient takes his dinner, which should consist simply of a mutton-chop, from which the fat and gristle has been removed, and which has been cut up fine, as if prepared for a child. For tea, the patients take milk alone, or porridge. At first, it is necessary to put these patients upon a strict diet, and it must not be expected that they will improve at the very first. Sometimes it will be a week or ten days before the patients are much better, but I have not yet seen any patients who did not begin to improve by the end of ten days. When improvement once begins it is very rapid, and the patients begin to gain in flesh, strength, and spirits. After one or two weeks the quantity of food which the patients take is increased. The time at which the increased quantity of food is taken is at dinner, preceding which the stomach has been washed out. The patient is allowed to eat beef, poultry, bread, and fruits. After a time, as the patient continues to improve, the stomach will not require washing out so frequently. Finally, it is no longer necessary to have the stomach washed. The patients cease to have any pain, do not vomit, and continue very well. By this time they have learned to use the stomach-pump, and, if intelligent, they will carry on the treatment themselves. At any time when they begin to have pain, they will wash out the stomach and put themselves on a strict diet. In this way the treatment is exceedingly satisfactory.

In the second set of cases, which are marked by vomiting from time to time, the indications are the same, namely, to do thoroughly what the patient incompletely accomplishes by vomiting, the emptying of the stomach. This is accomplished by the funnel or stomach-pump. As a rule, it is not necessary to wash out the stomach more than once a week, even at the commencement of the treatment, and it is not necessary to put these patients upon so strict a diet as the preceding. All that is necessary is to arrange their meals so that when the stomach is washed it will not be filled with solid food. In these cases, the results of treatment are satisfactory, but not so much so as in the preceding set of cases. In many cases the patients are not permanently cured, and it is necessary for them to continue to empty the stomach from time to time.

In the third set of cases, the treatment by emptying the stomach is utterly unsuccessful so far as a permanent cure is concerned. These patients do not get well. The stenosis is apt to become more and more marked ; the pylorus gets smaller and smaller, and although emptying the stomach makes the patient more comfortable, it does not cure the disease, or permanently relieve the symptoms. In most of the cases, unless you see them very late in the disease, you cannot at first be certain as to whether there is real stenosis of the pylorus from the physical signs. But you find this out very soon if you place the patient under treatment, for you will find that each time you empty the stomach, if stenosis does not exist, there will be less and less food found in it, for the stomach will gradually perform its function better, so that there will be less food to be removed by the pump. If there is stenosis of the pylorus, you will find every time you empty the stomach that there is a larger amount of partly digested, and some completely digested food, so far as stomach digestion is concerned. Instead of being called upon to empty the stomach less and less frequently you have to empty it more and more frequently. The stomach is not able to empty the food it contains through the pylorus, and hence every time you empty it you find in it a large amount of partly digested food.

LECTURE XXXI.

LESIONS OF THE STOMACH—CONTINUED.—PHLEGMONOUS AND TOXIC GASTRITIS.

Phlegmonous, or Suppurative Gastritis; Pathology, Occurrence, Symptoms, Treatment.—Toxic Gastritis; Etiology, Pathology, Prognosis, Symptoms, Treatment.

PHLEGMONOUS, OR SUPPURATIVE GASTRITIS.—*Gentlemen:* We come now to another inflammatory affection of the stomach, which is called by the name of *phlegmonous, or suppurative gastritis.*

Pathology.—In this disease the inflammatory process involves the connective-tissue coat of the stomach. It does not involve the mucous membrane or the muscular coat, except secondarily, but it begins in the layer of connective tissue between the mucous membrane and the muscular coat. The connective tissue becomes the seat of suppurative inflammation and there is a production of pus and fibrin. In some cases the process is diffuse, and the whole connective-tissue coat of the stomach becomes involved. In other cases the inflammation is circumscribed. It involves some particular part of the connective-tissue coat, having an area of from two to three inches in diameter, and when this is the case, the products of inflammation will only be found in this particular part.

When the inflammatory process is fairly developed, it involves, as a rule, the peritoneal coat of the stomach. This will be covered with fibrin and pus, and very frequently the local peritonitis will extend to the rest of the peritoneum, and the patients will have general peritonitis, this being secondary to the inflammation in the stomach. Again, there will be cases in which perforations through the mucous membrane of the stomach will be found, so that pus will escape into the cavity of the stomach.

Occurrence.—The disease is fortunately not very frequent. In New York, once in two or three years, a case is reported. For some reason the disease seems to occur more frequently in males than in females. All of the cases that have been recorded occurred in adult life. No cases, so far as I know, have occurred in children, and none in very old persons. As to the causation of this inflammatory process, we are totally ignorant.

Symptoms.—The symptoms come on suddenly, as a rule. A patient who has previously been in good health may be suddenly seized with vomiting, which will be very profuse, persistent, and violent. The patient will first vomit everything which his stomach contains, and then continue to make attempts at vomiting, bringing up a little mucus and fluid. In some cases which have been recorded, this vomiting has succeeded a too free indulgence in eating of indigestible food. The vomiting being first instituted to get rid of this mass, is afterward continued. Besides vomiting, the patients complain of marked pain and tenderness over the region of the stomach. The pain is very severe indeed, being like that of peritonitis. In addition to this, they have at times a febrile movement. When this is once developed and it is pretty fairly pronounced, the patients have a temperature of from 103° to 105°. Besides this, there is a very decided prostration of the patient, and by the end of twenty-four hours he will look as if he had been suffering from some severe constitutional disease. After this cerebral symptoms come on and the patient becomes stupid, and may be delirious, and finally die in a condition of collapse.

If there is general peritonitis added to the inflammation of the stomach, the additional local symptoms consequent upon this will be observed. There will be the pain and tenderness over the abdomen, common to peritonitis. The pulse is at first rapid and feeble, and it becomes more and more so toward the close of the disease. The temperature, I have said, is high at first, and continues thus until toward the end of the disease, when it may fall, as in peritonitis, and the patients may die in collapse with a comparatively low temperature.

Prognosis.—All of the cases that have been observed so far have proved fatal. When the inflammation is diffuse, patients usually die within a week from the commencement of the disease. But when it is circumscribed, the duration may be somewhat longer than this.

Treatment.—So far as treatment is concerned, we can do nothing except make the patients as comfortable as possible, and to accomplish this, they are to be treated as cases of acute general peritonitis. They are brought, as soon as possible, under the influence of opium, and kept under it while the disease is running its course. This is done for two reasons: first, to relieve the extreme pain, which can only be done in this way; and second, because the diagnosis is doubtful, there being always a possibility that the case is one of acute general peritonitis; so we adopt the treatment of the latter disease. This is the only disease with which it can be very well confounded, and in a very considerable number of cases the diagnosis is not made at all during the life of the patient. Many cases have been supposed to be acute peritonitis which showed, at the post-mortem, the lesion of the stomach.

TOXIC GASTRITIS.—We come now to that form of inflammation of the stomach which is due to the ingestion of poisons. This variety of gastritis is frequently called *toxic*.

Etiology.—The poisons which most commonly produce gastritis in this way are the mineral acids, sulphuric acid, nitric acid, and muriatic acid. Phosphoric acid also may produce this condition, although less frequently. It is also caused by some of the strong vegetable acids, such as strong oxalic acid, or acetic acid. Carbolic acid is also the cause in some cases. Strong alkalies are sometimes taken, as caustic soda. The preparations of arsenic, especially arsenious acid, and the arsenite of copper, may cause it, and less frequently Fowler's solution of arsenic. The bichloride of mercury will also excite gastritis in the same way. There are a number of other poisonous substances which may act in this way, but the ones I have enumerated are the most common.

Pathology.—The condition in which the stomach is found after death varies with the amount of poison which has been taken and its concentration. If the mineral acids, carbolic acid, or the strong alkalies are taken into the stomach in considerable quantities in a concentrated form, they will almost immediately destroy a part of the wall of the stomach, so that there will be a large perforation through it. More frequently only the mucous coating is destroyed, the process not extending through the cellular, muscular, and peritoneal coatings. If these poisons are taken in less quantities and are diluted, they will not destroy any considerable part of the wall of the stomach, although there will be death of the superficial parts of the mucous membrane. A general inflammation of the stomach will set in, which will usually be of the croupous variety, and the inflammation will be attended with the production of fibrin and pus in the substance and upon the surface of the mucous membrane.

The vegetable acids are, as a rule, not strong enough to exert a caustic effect upon the wall of the stomach. They simply excite an inflammation of the croupous variety, or occasionally of a catarrhal nature. If not in a concentrated form, they will simply set up a catarrhal gastritis. When arsenious acid, arsenite of copper, or corrosive sublimate are taken into the stomach in substance, after death it is commonly found that some of the poison is still in the stomach, adhering to the mucous membrane. Wherever it adheres in this way the membrane beneath it is partly destroyed, the poison having acted as a superficial caustic. Again, little spots of blackened mucous membrane are found in other parts of the stomach. These have been acted upon in the same way, but there is no longer any poison adhering to the surface. Around these spots are zones which have been the seat of croupous or catarrhal inflammation. The inflammatory process has extended, in many cases, to the connective tissue coating of the stomach, which may be infiltrated with pus and fibrin.

When the arsenite of copper has been taken, a considerable part of it is almost always found in the stomach. In New York, those who have taken arsenite of copper have taken it in large quantities, and for the reason that they are aware that even after they have vomited a good deal and the stomach-pump has been used, some of the poison will still adhere to the inside of the stomach. I know of no poison that is taken in such large quantities as arsenite of copper. If the patient lives some time after the ingestion of the poison, the inflammatory process will extend from the stomach to the small intestine, and this is especially the case when preparations of arsenic and corrosive sublimate have been taken. The patients not only have gastritis, but they have also enteritis. The inflammatory process in the intestine is usually catarrhal, and is accompanied with the production of a large amount of mucus. The small intestine is congested and its mucous coat is covered with a thick layer of mucus.

Prognosis.—If the patients recover from this disease, a certain number of them recover completely. The inflammatory process subsides, and the affected parts of the mucous membrane return to their normal condition. In other cases patients recover with a stomach which does not return to a natural state, but is permanently damaged. Where a considerable amount of mucous membrane has been destroyed, the loss is made up by cicatricial tissue, and the contraction of this deforms the stomach in a variety of ways. Under these circumstances the patients will continue to manifest gastric symptoms during the rest of their life.

Symptoms.—The symptoms of toxic gastritis vary with the character and strength of the poison. The regular symptoms are vomiting, pain, purging, cramps of some of the voluntary muscles, especially of those of the abdomen, and in the fatal cases, collapse. If the patients take a large amount of one of the mineral acids, or caustic alkalies, or carbolic acid, the most marked symptom is collapse. In the worst cases they hardly vomit at all, but die in a few hours in a state of collapse. If, however, the amount of one of these poisons is small and somewhat diluted, the patients will have vomiting and pain, the amount of prostration depending upon the amount of poison taken. If only a small amount of poison is taken, the prostration will not be very pronounced, and the vomiting and pain will be the most marked features. If the patients have taken one of the preparations of arsenic, or have taken corrosive sublimate, there is vomiting and pain, and after a little time purging, accompanied with more or less prostration, according to the amount of poison taken.

If the case is to prove fatal, these symptoms will continue and the patient will die collapsed at the end of a few hours, or several days. If, however, the amount of poison is not sufficient to cause death and it be promptly removed from the body, the gastritis will continue for some time after the patient's recovery from the immediate effects of the poison.

We will suppose a case of arsenic-poisoning occurring in a prostitute. The greater number of prostitutes in New York who take poison take a considerable amount of Paris-green. If the amount taken has not been large, and the stomach has been emptied soon afterward, the patient may not die. At first, she will complain of pain in the region of the stomach. She will not be able to take food by the mouth and it will have to be given by the rectum. After a few days the pain will seem to subside, and she will now be able to take small quantities of milk by the mouth without vomiting. This improvement will last for a few days and then the patient will begin to complain of pain, and reject food taken by the stomach. Not only this, but she will vomit a little blood. This second attack of the symptoms marks the time when the sloughs are separated from the mucous membrane, at which time there is a little bleeding from the granular tissue which has been formed. The symptoms coming on for the second time will last for several weeks, during which time the patient cannot take food into the stomach. The pain continues and there is vomiting of blood occasionally. After a time the symptoms subside, and the patient is first able to take fluid food, then solid food, and finally recovers altogether.

Treatment.—The treatment of toxic gastritis varies, of course, with the character of the poison which is taken. If one of the mineral acids has been ingested, alkaline solutions should be introduced into the stomach. The patients are caused to drink large quantities of solutions of magnesia, or carbonate of soda; or carbonate of potash, and to this is added either milk or some demulcent drink, the object being to dilute and neutralize the poison as much as possible. In addition to this, the pain is relieved by giving opium.

If it is oxalic acid that has been taken, solutions of lime are given with a view of diluting the poison. If it is one of the caustic alkalies, the patient is caused to drink large quantities of diluted acids, and to use demulcent drinks and milk, in the same way as before. If the patient has taken carbolic acid, a saccharated solution of lime is given, together with demulcent drinks, and opium used to relieve the pain.

If arsenic has been taken, the stomach is emptied, if possible, by means of the stomach-pump or stomach-tube, and the regular antidote, the hydrated peroxide of iron, in half-ounce doses, is given every five minutes.

If corrosive sublimate has been taken, the antidote for this is albumen, and usually the most convenient form is the white of an egg. The white of four eggs is taken and mixed with a quart of water, and the patient caused to drink this. But, before doing so, a trial should be made to empty the stomach. In cases of arsenic-poisoning which do not prove fatal but go on improving, one of the practical difficulties is the feeding of the patient. In a large number of instances these patients are totally unable to take any food by the mouth, even a teaspoonful of water will be vomited, and this will be kept up for a considerable length of time. So these patients have to be fed by nutritive enemata, and at the same time it is necessary to control the pain. In some this can be done with opium, but it must be remembered that one of the effects of arsenic-poisoning may be to set up a parenchymatous nephritis, in consequence of which the quantity of urine voided will be small. This being the case, it will be found that these patients are unusually susceptible to the constitutional effects of opium, so that an amount of opium which is not sufficient to control the pain may cause alarming symptoms. In view of this fact I have used, in two cases, iodoform for the relief of pain, bearing in mind the usefulness of this drug for the relief of the painful sensations of mucous membranes. In these two cases, the pain was relieved completely. I used, first, the iodoform in the shape of pills, but I found it did not answer. Patients objected to taking it in this form, stating that it caused them to vomit, so I gave it in solution, in five-grain doses, every three or four hours. Whether the remedy will answer in other cases as well, I do not know, but in the cases cited it relieved the pain, which we were unable to control by the use of opium.

From time to time, as the case progresses, a trial should be made to see whether the patient can take food by the stomach, for the patient cannot be nourished indefinitely by the rectum. A teaspoonful of milk may be given every hour or two. If the patient is able to retain it, its administration should be continued until the period of secondary inflammation is reached, due to the separation of the slough; then it is commonly necessary to return to the use of nutritive enemata for a time, after which the patient can be fed by the stomach again. The suppression of urine is sometimes so marked that it may be necessary to give the patient hot-air baths. Only five or six ounces of urine may be passed during the twenty-four hours, which always means that the patient is in a dangerous condition, and I think it would be proper, in these cases, to give a hot-air bath. These constitute the ordinary measures employed in the treatment of toxic gastritis.

LECTURE XXXII.

LESIONS OF THE STOMACH—CONTINUED.—ULCER OF THE STOMACH.

Chronic Perforating Ulcer of the Stomach.—Course of the Disease, Etiology, Symptoms, Prognosis, Exceptional Cases, Duration, Treatment.

CHRONIC PERFORATING ULCER OF THE STOMACH.—*Gentlemen:* We come to-day to the consideration of chronic perforating ulcer of the stomach. These ulcers, which we find occasionally in the stomach, are commonly either of a circular or elliptical shape. Occasionally two or more ulcers will become joined, and the shape of the whole ulcer will be quite irregular. At times they will form a sort of girdle around the pyloric end of the stomach. Such ulcers vary in size very considerably. Some are no larger than half a pea, while others will be half an inch, an inch, or two inches in diameter. The largest ones are those formed by the fusion of several smaller ones.

The appearance of these ulcers is peculiar. They do not look like ordinary ulcers of the skin or mucous membrane. But in some part of the stomach it will be observed that there is simply a loss of substance. It appears as if a circular piece of mucous membrane had been punched out and removed; there is no granular tissue, no congestion of the parts, and in fact no evidence whatever of the existence of an inflammatory process. Such a loss of substance may involve only the mucous membrane, or it may extend to the connective-tissue coat, or deeper, and involve both the muscular and peritoneal coats. The size of the ulcer decreases as its depth increases; thus, if it involves the entire thickness of the wall of the stomach, its circumference will be greater in the mucous membrane than in the peritoneum. In a large number of instances the edges of the ulcer are not at all thickened. If examined with a microscope, no granular tissue or any number of new cells will be found, only the normal anatomical elements which compose the wall of the stomach in a degenerated condition, will be found. The whole process is a destructive one, and not an ordinary inflammation. Occasionally the edges of the ulcers are thickened, and this will be found to be due to the growth of new connective tissue which contains a considerable number of cells.

The number of ulcers which may be found in a single stomach varies from one to five. As a rule, we only find a single ulcer. Occasionally we will find in a single stomach one or more ulcers which have healed, while there are others which are apparently still perforating.

Their position is usually on the posterior wall of the stomach, near the pyloric end. It is estimated that nearly forty-three per cent. of all ulcers of the stomach have been upon the posterior wall; that twenty-seven per cent. have been upon the lesser curvature of the stomach, and that sixteen per cent. have been situated at the pylorus. Upon the anterior wall and greater curvature of the stomach we find these ulcers much less frequently. If the ulcers are not very large and have not perforated very deeply, it is possible for them to heal without producing any deformity of the stomach, the lost substance being replaced by new connective tissue which does not contract. If, however, the ulcers are of considerable size and have perforated pretty deeply into the wall of the stomach, should the patient get well the ulcers cicatrize and the contraction of the cicatrices alters the shape of the stomach. If the ulcer has perforated through to the peritoneal coat in a certain number of cases, an inflammatory process will be set up in this coat of the stomach, and, as a result, this particular part will become adherent to the adjacent viscera. The viscus to which the stomach most frequently becomes adherent is the pancreas, but it may also adhere to the liver or any of the surrounding parts. When the stomach has thus become adherent, the destructive process may extend into the adjacent viscus, as the liver or pancreas, and a cavity may be hollowed out in it which communicates with that of the stomach.

In other cases, no such adhesions are formed, and when the ulcer perforates, it does so suddenly. The contents of the stomach are discharged at once into the peritoneal cavity, and, as a result, there is death of the patient within a few hours; or else there is set up a peritonitis which usually terminates fatally. In still other cases, some adhesions are formed, but these are not sufficient to close the opening through the wall of the stomach. When this occurs, the contents of the stomach slowly escape into the peritoneal cavity. This sets up a local peritonitis, which goes on to develop the products of inflammation in the neighborhood of the ulcer in the stomach. Not only may a chronic peritonitis be set up in this way, but abscesses may be formed —collections of pus which may be shut in by adhesions. These may become larger and larger, and as they do, they involve the soft parts. They may extend to the adjacent viscera, into the liver or pancreas, or to the anterior abdominal wall, or any of the surrounding soft parts. Ulcers in the stomach are usually accompanied with a certain amount of chronic catarrhal gastritis, and this always has a share in producing the symptoms of which the patient complains. In ordinary cases of ulcers of the stomach, the symptoms manifested are due partly to the presence of the ulcer and partly to the chronic gastritis which accompanies it.

This lesion of the stomach is tolerably frequent. It is twice as frequent in females as in males. In males it occurs at all ages after early childhood. It has been observed in persons between the ages of eight and eighty years. The disease, however, has a preference for certain ages. In women, the larger number of cases occur between the ages of fourteen and twenty years. In males, the disease, for the most part, occurs in persons rather older, or between the ages of twenty and thirty years.

Etiology.—It occurs without any known cause. It is found in some persons suffering from pulmonary phthisis, in chlorotic women, and in persons who have been badly nourished for a considerable length of time, but a number of cases occur without the coexistence of another disease or any known cause.

It has always been a question as to how these ulcers are formed. The ordinary explanation which is given of their formation is that a certain part of the wall of the stomach is deprived of its vascular supply, and it may be thus deprived, either by the obstruction of one of the arteries of the stomach (by an embolus), or by fatty degeneration, or there may be a narrowing of the calibre of a number of the small arteries of the stomach, or there may be extravasation of blood into the tissue of the stomach. When the nutrition of a part of the wall of the stomach has been shut off in this way, it ceases to be alive. When the stomach-tissue has become dead, it may be acted upon by the gastric juice in the same way that other dead animal matter is acted upon by the gastric juice. As a result, there is a loss of substance and the formation of ulcers. This is the common explanation of the formation of these ulcers—that they are formed by the action of the gastric juice upon a part of the wall of the stomach which has lost its vitality. This explanation, although it is the best we have, is not by any means satisfactory. There are many objections to it, and many reasons for its not being satisfactory, but still, it is the most probable explanation which has yet been offered.

Symptoms.—The symptoms produced by ulcer of the stomach are commonly pretty well marked. The most prominent of these is pain, which is almost constant. There are, however, cases of ulcer of the stomach in which the pain is not a pronounced feature. Pain is usually referred to some point over the stomach, either to the pyloric end or to some part of the anterior wall of the stomach. In some instances, it is described as a continuous, dull pain, which is increased upon pressure, and by the movements of the patient, as well as by the ingestion of food. In other cases, the pain comes on in the form of sharp attacks. There will be a period during which the patient will have very little pain indeed, after which he will have sudden attacks of very severe pain, resembling those of neuralgia, which last for a few hours and then pass off, to return at another time. The tenderness upon pressure is deep, rather than superficial, that is to say, no pain is experienced by simply applying the hands to the surface of the abdomen, but when pressure is exerted slowly and steadily over the stomach considerable pain is caused.

In the larger number of cases, vomiting is a frequent symptom. There are cases, however, in which there is no vomiting at any period of the disease. The frequency of the vomiting varies very much indeed. Some patients will vomit several times during the twenty-four hours. Others will vomit once a day, or once in two or three days, or once a week. Sometimes the vomiting will only occur after the ingestion of food. In other cases, however, the vomiting is spontaneous: the patients will vomit whether they have taken food or not. The matters vomited are the food which has been taken into the stomach, mucus, and a certain amount of yellow, frothy fluid. In the vomited matter we very often find sarcina ventriculi, and in addition the patients will occasionally vomit blood. The vomiting of blood occurs in about one-fourth of the cases. It is important for you to remember the relative frequency with which these patients vomit blood, for it is, perhaps, too much of a habit to expect vomiting of blood in all cases of ulcers of the stomach. The blood which is vomited is sometimes in the form of a dark, grumous fluid, which does not look like blood, but very much like coffee-grounds, and, owing to this, it is most commonly called the coffee-ground vomit. It is simply blood that has been acted upon by the contents of the stomach and the gastric juice. In other cases the vomited matter will be streaked with blood. Again, at times, these patients may vomit pure blood in small quantities, and occasionally in very large quantities. In the very worst cases, patients will vomit cupfuls of pure blood, and it is even possible for patients to bleed to death from such an ulcer of the stomach. Death from this cause occurs in about four per cent. of all the cases of ulcer of the stomach.

Besides these marked symptoms of pain, vomiting, and vomiting of blood with emaciation, we ordinarily have dyspeptic symptoms, such as I have already enumerated under the head of gastric dyspepsia. The number and severity of these depend upon the amount of gastritis which accompanies the ulcer. The general health of the patients is, as a rule, depreciated, and they become emaciated, anæmic, and weak. The gastric pains cause the face to have a peculiar, anxious and haggard appearance.

Prognosis.—The terminations of ulcer of the stomach are various. About one-half of all the cases get well. The ulcer cicatrizes and the patients recover completely. In about thirteen per cent. of the cases, the patients die from perforation of the stomach by the ulcer. There may be, suddenly, a large perforation of the ulcer into the peritoneal cavity, and when this is the case patients may die in one of two ways. Some seem to die from the shock caused by the entrance of the contents of the stomach into the peritoneal cavity, and they at once pass into a condition of collapse. The skin is cold, the pulse feeble, and the patients die at the end of a few hours. In other cases, the patients recover from the immediate effects of the shock, but the contents of the stomach set up a general peritonitis and the patients go on to suffer from all the symptoms of this. Patients thus affected almost invariably die, but they may live as long as a week. In other cases, patients may continue to suffer from the results of partial perforation of the stomach from the symptoms of chronic localized peritonitis, or from those due to the formation of abscesses in the peritoneal cavity. As I have already said, these abscesses may perforate into the viscera or into the abdominal wall. When they perforate the abdominal wall, the pus may be discharged externally through the skin. These cases usually prove fatal, although the patients may continue to live for quite a long time, but sooner or later they are apt to die from chronic peritonitis and the presence of pus in the peritoneal cavity. In other cases, the ulcers may cicatrize, but they do so in such a way as to produce deformity of the stomach, and the result of this is, that either the whole cavity of the stomach is smaller than it should be, or else, if the contraction is near the pyloric end of the stomach, the stomach becomes dilated above the point of the contraction. When dilated in this way patients suffer from the ordinary symptoms of dilatation of the stomach. If the stomach is contracted, patients will, as a rule, suffer from vomiting and a certain amount of pain in the region of the stomach.

The larger number of fatal cases result from gradual starvation. Patients are able to take but very little food into their stomachs, and consequently they become weaker and weaker, more and more emaciated, and finally die from starvation. There are also some who die from intercurrent disease, as phthisis or pneumonia.

Exceptional Cases of Ulcer of the Stomach.—Besides these regular cases of ulcer of the stomach, there are, unfortunately, some irregular ones which do not manifest the characteristic symptoms of which we have been speaking. The symptomatology of these irregular cases can, however, be summed up in a very few words. In the first place, some of them give rise to no symptoms whatever. Such patients die from some other disease, and at the autopsy we find an ulcer of the stomach which has existed for some time. In other cases, the ulcer only gives rise to a single symptom, and that is the death of the patient. A patient who has previously been in good health will suddenly pass into a condition of collapse, the collapse being due to the sudden perforation of the stomach by the ulcer which has previously given no symptoms whatever. Some of the patients, who thus suddenly go into a condition of collapse, die in a few hours from shock. In others, a peritonitis is set up and the patient continues to suffer from the effects of this, the peritonitis usually proving fatal. It is important to bear in mind that such a perforation is one of the causes of sudden peritonitis, and it may occur in a person who has not manifested any of the symptoms of ulcer of the stomach. It is often difficult to distinguish these cases from general peritonitis, due to inflammation of the vermiform appendix. You may, perhaps, think strange of this statement, but, as a matter of fact, it is sometimes difficult to determine which of the two causes has given rise to the peritonitis. There are cases of inflammation of the vermiform appendix in which pain is never referred to this locality, but will be referred to the whole of the abdomen, and when there has been no previous symptom of ulcer of the stomach, we may be at a loss to know the cause of the peritonitis.

Duration.—The duration of ulcers of the stomach may be for a longer or shorter time. Some patients manifest the symptoms for several months, and the larger number of them for a year. Most of the cases that become convalescent require a year for the disease to run its course. The continuance of the disease, in other cases, may be for a period of twenty or thirty years, during all of which time the patients will suffer from symptoms of ulcer of the stomach. In these protracted cases, it is always difficult to tell whether the symptoms are due to the existence of one or several ulcers, and this can only be surely determined at the post-mortem. It is also difficult to say how many of the symptoms are due to the chronic gastritis which accompanies the ulcer, or ulcers.

Treatment.—In the treatment of ulcers of the stomach, we have, usually, two indications to fulfil. First, to nourish the patient, and secondly, to alleviate the symptoms from which he is suffering. Pain and vomiting, the most prominent of the symptoms, are often best treated simply by the regulation of the patient's food; and especially is this the plan to be adopted in the case of those patients in whom the pain and the vomiting are regularly induced by the ingestion of food. There are two ways by which we nourish these patients. We try to feed them by the stomach with food that can be easily digested, that is, that kind of food which requires only a small amount of stomach digestion; or else we give the stomach complete rest and feed them by nutritive enemata. In feeding them by the stomach, we use three articles of diet, viz., milk, beef-tea, or the juice of beef, or meat that has been partly digested. We give these patients small quanties of one or another of these articles of diet. We do not let them take a tumbler of milk at one time, but half an ounce, and that pretty frequently. The best preparation of partly digested meat is that of Liebig, the formula by which it is prepared being given by a German physician of that name. It is prepared in this city by Mr. Hoffman, druggist, at the corner of Sixth avenue and Forty-sixth street, who constantly keeps a supply of it. It is a very useful preparation of meat to employ in the treatment of many of these cases. You give a teaspoonful of it every two or three hours. The beef-tea may be prepared in the ordinary way, or you may use the juice which has been squeezed from meat. In this way you can nourish these patients very well for a considerable length of time.

There are other cases, however, in which the stomach will not tolerate such fluid food. Even if it is given in small quantities, it will bring on pain and vomiting. Under such circumstances, you have to resort to the use of nutritive enemata, which are to be administered two or three times every twenty-four hours. Each time before their administration you give a small enema of tepid water, in order to remove from the lower part of the rectum whatever fæces are present. After this, the nutritive enema is given in quantities from half an ounce to two ounces, according to the patient's tolerance. The substances which we use for this purpose are the same as those I have spoken of as being employed by the mouth. In many cases enemata composed entirely of milk, or beef-tea, or the yolks of eggs, will not be retained, and will be expelled in a few minutes. But, by the addition of a small amount of brandy and opium, these enemata are usually retained. You can feed a patient in this way for a considerable length of time: they have thus been kept alive for many weeks.

There are certain drugs that seem to have some effect upon the symptoms from which these patients suffer. The most effective is nitrate of silver, in pill form, and in doses of one-fourth of a grain given three times a day. This drug seems to diminish the pain and vomiting. In its administration you must avoid continuing its use for a great length of time, or else the characteristic discoloration of the skin due to its use will obtain. The other drugs which may be used are the bicarbonates of soda and potash, the oxalate of cerium, hydrocyanic acid, and the preparations of bismuth, all of which apparently do good, not by any effect which they have upon the ulcer, but by their effect upon the chronic gastritis which always accompanies ulcer of the stomach. It should always be borne in mind that a considerable number of the symptoms in these cases are due to the accompanying gastritis, and that the value of drugs depends upon the effects which they have upon the gastritis.

When pain exists only after the ingestion of food, it may be relieved by feeding the patient by the stomach with small quantities of properly prepared food, or by feeding the patient per rectum. Pain sometimes exists independently of the ingestion of food, and no matter whether the patients take food or not, they continue to have severe pain, and it is so severe that it really constitutes an evil, for the patients lose flesh and strength in consequence thereof. Sometimes it is impossible to nourish the patient by means of rectal enemata. Hence, it is always a question of a great deal of importance as to how the pain can best be removed. It is the one symptom of which the patients complain most, and it has a very bad effect upon their general health. When nitrate of silver has failed to relieve the pain, you are always brought face to face with the question whether it is best to resort to the use of opium. The pain can always be relieved by this drug, and not infrequently we are absolutely driven to its use on account of the excessive pain which these patients suffer, but its administration should never be begun in ulcer of the stomach without a clear understanding of what is being done. It should be remembered that the disease lasts for months, sometimes for years, and when a patient has once begun the use of opium he will not be able to leave it off until the ulcer has cicatrized. Not only this, but the drug has to be used in increased doses, for the amount which at first relieved the pain will be ineffectual later on in the disease, so that in a short time these patients will be fairly established in the opium-habit. They are no longer comfortable without it; they feel the need of it, not only for the relief of the pain in the region of the stomach, but also for the relief of the restlessness and discomfort which those who have once acquired the opium-habit experience when they are no longer under its influence. So that when the ulcer of the stomach has disappeared, you have before you a patient to treat for the opium-habit, and this is always a difficult matter.

There is still another procedure which is of value for the relief of pain in some cases. This consists in the formation of a nitric acid issue over the epigastric region. To do this, you cut from a card a circular piece about the size of a twenty-five-cent coin, soak it in nitric acid and lay it upon the skin in the epigastrium and allow it to remain in this position for ten minutes; it is then removed, the part wiped and covered with a piece of adhesive plaster. In the course of a few days a slough will come away and there will be a little circular ulcer which will remain open as long as you keep it covered with adhesive plaster. When you wish it to heal, all that is necessary is to remove the adhesive plaster and it will soon disappear. Such a nitric acid issue will in some cases relieve the pain completely, or to such an extent that it will not be necessary to resort to the use of opium.

Hence, for the relief of pain, you try, first, the effect of a regular diet; if this fails, you are to feed the patient by the rectum. If this does not answer the purpose, the use of nitrate of silver should be tried; if this fails, nitric acid blister; and if this does not succeed, there is no other recourse except to opium.

If blood is vomited in such quantities as to render the patient feeble or anæmic, it may be necessary to do something for the relief of this symptom. For this purpose there are two drugs, ergot and gallic acid, which are perhaps more efficacious than any other, and the advantage to be obtained from their use will depend upon the freedom with which they are administered. If it is desired to arrest the hemorrhage, ergot may be given by the mouth in the form of the fluid extract. As a rule it cannot be administered hypodermically in sufficient quantities to be of very great service. If a patient is bleeding pretty freely, it may be given in drachm doses every two, three, or four hours, according to the amount of bleeding. There need be no hesitation in giving the drug thus freely, for it is not likely to produce symptoms of ergot-poisoning. I have given drachm doses of the fluid extract of ergot every two hours for a week in cases of pulmonary hemorrhage, without the patient developing symptoms of ergot-poisoning. In the same way gallic acid should be given freely. It can only be administered by the mouth and in the form of powder or pill, or, it may be given in a mixture in conjunction with sulphuric acid. As a rule, it is best used in five-grain pills, at first every hour, and then every two, three, or four hours, according to the profuseness of the hemorrhag If the hemorrhage is not free enough to suddenly cause the death the patient, it may be controlled, as a rule, in a short time, by the u of one or the other of these drugs.

LECTURE XXXIII.

LESIONS OF THE STOMACH—CONCLUDED.— CANCER OF THE STOMACH.

Cancer of the Stomach; Varieties, Pathology, Site, Course of the Disease, Etiology, Symptoms, Physical Signs, Exceptional Cases, Prognosis, Treatment, Differential Diagnosis.

CANCER OF THE STOMACH.—*Varieties.*—*Gentlemen:* We come now to speak of cancer of the stomach. In the stomach are found all the different anatomical varieties of cancer, but there are three of these which are much more common than any of the others; these are the only varieties that it is necessary for us to take into consideration. Two of these varieties of cancer present exactly the same appearance to the naked eye, but in their minute anatomy they are different. The third variety differs from the other two both in its appearance and minute structure. These three varieties of cancer we may call by the names of simple carcinoma, epithelial carcinoma, and colloid carcinoma.

Pathology.—In simple carcinoma, the growth begins in the layer of connective tissue immediately beneath the mucous membrane of the stomach. The growth is formed of irregular spaces filled with polygonal cells. Between these are stroma or cells, the proportion of stroma and cells varying. If the cells are very numerous, and the stroma scanty, the cancer will be soft, and hence it is sometimes called *soft cancer.*

In the epithelial variety of cancer of the stomach, the new growth is formed of tubules lined with cells resembling the cylindrical tubules of the stomach, and between these the stroma is situated.

Both of these varieties of cancer, although they differ in minute structure, have exactly the same gross appearances. They also begin in the same place, namely, in the layer of connective tissue immediately beneath the mucous membrane. Beginning here, however, they may grow in several different ways. In many cases, they grow inward toward the cavity of the stomach, so that flattened tumors are formed which project into it. These are very apt indeed to ulcerate at their centres. The ulceration may extend quite deeply, so as to destroy the new-growth and even invade the wall of the stomach beneath it. When the ulcerative process has made considerable progress, the appearance presented is no longer that of a tumor, but of an ulcer with thick, overhanging edges. In other cases, there are polypoid tumors, which may be as large as a hen's egg, or that of a turkey, or even as large as a small orange.

If these tumors are situated at the pylorus they will act as a sort of valve to block the pyloric end of the stomach. In some cases, instead of there being a tumor formed, there is a diffuse infiltration of the wall of the stomach, and the growth, instead of extending inward, and forming a circumscribed tumor, extends laterally, and infiltrates the walls of the stomach. Such an infiltration may be confined to the pylorus, or it may extend over a very large part of the wall of the stomach. In other cases, we simply find a diffuse thickening, involving a larger or smaller area of the wall of the stomach. These are the three ways in which this form of cancer may grow.

Colloid cancer of the stomach has the same structure as colloid cancer occurring elsewhere in the body. There is a stroma of connective tissue, and in this are situated irregular cavities filled with a gelatinous material which, when fresh, looks like clear, transparent jelly, and in which are embedded polygonal nucleated cells. This form of cancer begins like the other two, in the connective-tissue coat of the stomach, but its tendency is to extend outward rather than inward, and infiltrate the muscular coat. The diffuse thickenings produced in this way are very marked indeed. The wall of the stomach, although retaining its regular shape, may be one, two, three, or four inches in thickness, which is due, partly, to its infiltration with the new growth, and partly to the new growth of connective and muscular tissue.

Site.—All these three forms of cancer of the stomach are found much more frequently at the pyloric end, and usually upon the lesser curvature and posterior wall. If the growth takes on a diffuse form, the whole stomach may be involved by it. In some cases the size of the flattened tumor is so great that it will cover the whole of the lesser curvature and posterior wall of the stomach. Sometimes the new growth is not situated at the pyloric, but at the cardiac end of the stomach. When this happens the growth may extend upward and involve the œsophagus. When it is situated at the pyloric end it very seldom advances beyond this point.

Course of the Disease.—The development of these tumors is very likely to be attended with inflammation of the peritoneal coat of the stomach in the neighborhood of the new growth, resulting in the formation of adhesions between the wall of the stomach and the adjacent viscera. In consequence of such adhesions, if the cancer ulcerates, the process may extend so deeply as to give rise to perforation of the stomach; but this does not usually take place into the abdominal cavity, for the bottom of the ulcer is closed by the adjacent viscera which have become adherent as a result of the localized peritonitis. In about forty-eight per cent. of the cases of cancer of the stomach, secondary tumors having a cancerous structure are formed in other parts of the body. The liver is the organ in which these secondary tumors most frequently make their appearance. They usually grow in the form of circumscribed nodules scattered through the substance of the liver. They may be large or small. Occasionally the liver itself will be very much enlarged, and when this happens, it is of great assistance in making the diagnosis of the condition of the stomach by means of palpation. In other cases the liver will remain of its natural size, and in still other cases it will be actually diminished, although it contains these nodules of new growth. Next in frequency to the liver as the site of secondary cancerous growths are the lymphatic glands, especially those which are situated in the immediate vicinity of the cancer of the stomach. When these glands become infiltrated by the new growth they are very likely to become adherent to the wall of the stomach, and directly opposite the position which the cancerous tumor occupies in that organ. Becoming adherent in this way the new growth in the wall of the stomach and that in the lymphatic glands may form a single tumor. This happens in a very large number of cases. Curiously enough, in some cases of cancer of the stomach the lymphatic glands of the neck are involved, especially those upon its anterior part. What the reason of this is I do not know, but such is the case. The other lymphatic glands of the body are less likely to be affected. Besides the occurrence of secondary growth in the liver and lymphatic glands, tumors may form in almost any part of the body, but this takes place much less frequently.

Etiology.—The disease seems to occur with about equal frequency in the two sexes, the average age being fifty-one years in males and forty years in females. The maximum liability to the disease, in both sexes, is between the ages of sixty and seventy. Cancer of the stomach may be almost excluded in individuals under thirty years of age; very seldom indeed does it occur in younger persons.

As regards the causation of cancer of the stomach, we have no more knowledge than we have in regard to the causation of cancer elsewhere in the body. We have to remember that it is a disease which is very common; so common, indeed, is it, that one, seeing many patients, cannot fail to have a number of examples of it come under his observation every year. It is a disease for which physicians should be constantly on the watch.

Symptoms—The appetite of patients suffering from cancer in the stomach is diminished, or absent altogether. Nausea and vomiting are very common symptoms; it is estimated that they occur in about eighty-seven per cent. of all the cases. Vomiting, however, is not so common a symptom in cancer as in ulcer of the stomach. There is a great deal of difference among patients with cancer of the stomach as to the way in which they vomit, and as to the time at which this occurs. In some cases it will be one of the first symptoms that will attract the attention of the patient. It will come on either very soon after eating, or one or two hours afterward, and may continue throughout the entire course of the disease. As the disease progresses, vomiting after eating becomes a more constant symptom. In other cases, patients do not vomit every time they eat, or every day. They may vomit only once a week, or once in ten days, but when they do vomit, it is in considerable quantities. The vomited matter consists partly of a yellowish and brownish fluid mixed with the food. In other cases patients will vomit at the beginning of the disease, and after a short time, for some reason they will stop, though the cancerous growth continues to develop. Owing to the subsidence of this symptom, patients sometimes think they are much better. In still other cases, the vomiting will not be present until within the last two weeks of the disease. Again, vomiting may be even more irregular, there being periods of weeks during which patients will vomit very frequently; then they will cease to vomit for weeks, after which they will begin to vomit again, and so on. It is somewhat important to remember the different manifestations of the symptoms of vomiting, for it is one of the symptoms which is regularly associated with cancer of the stomach, although, in exceptional cases, it may not be present. In about half of the cases, or more accurately forty-two per cent. of them, vomiting of blood is present. This symptom is more common in cancer than in ulcer of the stomach. In ulcer of the stomach vomiting of blood occurs in about one-fourth of the cases. As in ulcer of the stomach, the character of the blood vomited varies, sometimes it being of a dark, grumous, coffee-ground color, and sometimes clear blood. It may also be vomited in large or small quantities. Generally speaking, blood is vomited in smaller quantities in cancer of the stomach than in ulcer of the stomach, patients vomiting a little from day to day, but really a great deal in the aggregate.

Pain is a very constant symptom in cancer of the stomach. It is estimated to be present in ninety-two per cent. of the cases, and it is, if anything, more severe and constant than in ulcer of the stomach, and has much the same character as in this latter affection. In some cases, it is only present after eating; in others, it is present all the time. In the later stages of the disease, pain is as a rule constant. It is sometimes a dull, continuous pain; at other times a very sharp and severe one, beginning over the site of the cancer, and radiating from this in different directions.

Physical Signs.—In about eighty per cent. of the cases of cancer of the stomach a tumor can be appreciated by palpation through the abdominal walls at some time during the course of the disease. You must not, however, expect to be able to make out the existence of a tumor in the majority of cases that come to you for treatment, for, as a rule, sufficient time will not have elapsed for its development.

In examining for these tumors you will have to remember the part of the stomach where they are situated. If located near the œsophageal end, you cannot make out the existence of any tumor by palpation. The tumors which may be made out by palpation are those situated at the pyloric end of the stomach and upon its greater curvature, and it is here where most of these new growths are to be found. The pyloric end of the stomach, under normal circumstances, is situated beneath the lower border of the liver and is covered by it, so that, it is impossible to feel it in a healthy person. When, however, there is a tumor of the pylorus, the rule is, that this part of the stomach is displaced downward to some extent, which makes it possible to feel the tumor through the abdominal wall if it is of sufficient size, for the pyloric end of the stomach is now below the lower border of the liver. The point at which these tumors are most frequently felt is in the lower part of the epigastric region, and when cancer of the stomach is suspected, it is here that we first palpate in order to determine if possible the existence of a new growth. Sometimes, instead of finding a tumor at this point, the dislocation of the pylorus will be in some other situation; it may be still lower and to the right, or far over on the left side.

If there is diffuse infiltration of the wall of the stomach, and it involves the anterior wall and greater curvature, a tumor of considerable size may be felt, which will give you an idea of the contour of the walls of the stomach. Sometimes, the lymphatic glands in the neighborhood of the stomach are infiltrated with a new growth, and when this happens they become adherent and add to the size of the tumor, which makes it more easy to map out by palpation. Generally speaking, the tumor which is found in cancer of the stomach is movable. It may be moved by the hand, and if the patient breathes deeply, the tumor will move downward and upward with the diaphragm, and these movements can be felt by the hand. Sometimes, however, the cancerous tumor is not movable. This occurs when there are a number of lymphatic glands between the stomach and vertebral column which are infiltrated with the new growths. These become adherent to the stomach and vertebral column. Such a tumor it is very difficult indeed to distinguish from examples of another set of tumors which grow from behind the peritoneum.

Patients may suffer from an irregular febrile movement, which possibly may depend upon the local peritonitis, although we are not at all certain as to the cause of it, but in some cases the thermometer will mark an elevation in the temperature from time to time, without there being any change in the patient, so far as can be observed.

The bowels are sometimes constipated, sometimes loose. They are especially apt to be loose toward the close of the disease. If the hemorrhage is pretty free, some of the blood will pass out of the stomach into the intestine, so that you will find in the fæces a certain amount of blackened blood. Some patients become jaundiced to a very considerable extent, and this happens when there are secondary new growths in the liver which have lasted some time. The patients become feeble, cachectic, and anæmic. The skin assumes a peculiar white color which is quite characteristic, and of some help in making a diagnosis. It is possible for patients with ulcer of the stomach to have nearly the same color as those with cancer of the stomach. Unfortunately, however, this peculiar appearance of the skin does not come on until late in the disease, when the patients are approaching its close and the diagnosis has been made some time previous.

After patients have become quite anæmic, they not infrequently develop dropsy. Their feet and legs may become œdematous and there may be serum in the peritoneal cavity. This condition seems to depend upon the anæmia. These are the ordinary symptoms of cancer of the stomach.

Exceptional Cases.—As in ulcer, so in cancer of the stomach there are exceptional cases. There are patients in whom we find none of these regular symptoms until a short time before death. A patient may have cancer of the stomach for a number of months, during which time there may be no vomiting, no pain, no vomiting of blood, no dyspeptic symptoms, no cachexia, no loss of flesh or strength; and after lasting all this time without developing any symptoms, the patient will suddenly begin to manifest the symptoms belonging to this condition; and there are still other patients who have no symptoms whatever.

Prognosis.—The prognosis of cancer of the stomach is uniform; it is always fatal, and the rule is that patients die within a year from the time the diagnosis is fairly made out. If you see a patient with cancer of the stomach and are able to make a positive diagnosis, you will be safe in saying to his friends that he will die within a year.

Treatment.—As regards the treatment, the only indication is to make the patients as comfortable as possible, and the two symptoms which you have to relieve are vomiting and pain. As a rule, these patients may be fed by the mouth, the stomach not being as intolerant in cancer as in ulcer of the stomach. Milk, beef-tea, the yolk of eggs, and other similar articles of diet may be taken into the stomach, and not infrequently patients will be able to eat ordinary articles of diet. It is rarely necessary to nourish these patients by the rectum, as we are so frequently obliged to do in ulcer of the stomach. Pain is to be relieved by the use of opium, and the rule for giving it is exactly opposite to that which should guide you in its use in ulcer of the stomach. In cases of ulcer of the stomach the use of opium is avoided as long as possible on account of the liability of the patient to recover from the disease with the opium-habit. In cancer, on the other hand, we begin its use as soon as it is necessary to control the pain, and we do so because the duration of the disease is limited, and it is a matter of no consequence whether the patient acquires the opium-habit or not, so that opium should be given in sufficient quantities to control the pain. For this purpose opium is administered in the form of morphine and hypodermically in small doses, in which way it controls the pain more efficiently than when given by the mouth, and causes less disturbance to the general condition of the patient.

Differential Diagnosis.—The two diseases with which cancer of the stomach is most likely to be confounded are ulcer of the stomach and chronic gastritis. The points upon which we are to rely to distinguish it from ulcer of the stomach are : the age of the patient, the way in which the patient vomits, the character of the pain from which he suffers, the more or less rapid depreciation in his general health, his strength, and the presence or absence of a tumor. To distinguish it from chronic gastritis, we can usually rely upon the difference in the severity of the symptoms. Patients with chronic gastritis, like those with cancer of the stomach, may suffer from vomiting, pain, and emaciation. They may vomit blood from time to time. They may even become somewhat cachectic ; but none of these symptoms are, as a rule, as marked in cases of chronic gastritis as in those of cancer of the stomach. Of course, the farther advanced the cancer is at the time when you see the patient, so much the easier will be the diagnosis ; for if no tumor is present the diagnosis will be much more difficult, and one has fairly to admit that there are cases in which a diagnosis cannot be made for a considerable length of time.

LECTURE XXXIV.

DISEASES OF THE INTESTINES.—INTESTINAL DYSPEPSIA.—CHOLERA MORBUS.

Intestinal Dyspepsia; Occurrence, Symptoms, Treatment.—Acute Catarrhal Inflammation of the Small Intestine, or Cholera Morbus; Pathology, Symptoms, Differential Diagnosis, Treatment, Prognosis.

INTESTINAL DYSPEPSIA.—*Gentlemen :* We come now to the consideration of some of the diseases of the small intestine. The small intestine, like the stomach, takes a share in the digestive process, and like that organ its share is a double one. Certain parts of the food are digested in the intestinal canal, and certain parts are absorbed, both processes going on at the same time. In the small intestine, as in the stomach, there may be a functional derangement without the existence of any organic lesion. Such a functional derangement we may call by the name of *intestinal dyspepsia*, to distinguish it from gastric dyspepsia.

Occurrence.—Intestinal dyspepsia may exist either alone or in company with gastric dyspepsia, or functional derangements of the liver, or of the large intestine. The cases which are the easiest to diagnosticate are those in which no such complications exist.

Symptoms.—Most frequently patients suffer from pain which is of a peculiar character. It is not usually like that of gastric dyspepsia, or ulcer, or cancer of the stomach. It is not commonly very circumscribed, but extends over a considerable area, and may be referred to any part of the abdomen, although it is most frequently referred to the upper part. The pain is not usually a sharp, neuralgic one, but of a dull and continuous character, and is at no time unbearable, although it annoys the patient to a considerable extent. It may come on at any time of the day, having no relation whatever to the ingestion of food, but not infrequently patients suffer pain at some particular part of the twenty-four hours, which is usually during the afternoon. The pain is not developed suddenly, as in colic, but is at first dull, and gradually becomes a little more severe. Its character is somewhat different from any other abdominal pain with which patients are afflicted.

If there are disturbances of the stomach there will be nausea and vomiting, but if the functions of that organ are properly performed these symptoms will be absent. There will be no change in the appetite, the patient being able to eat as much food as he desires. He will not be depressed by the ingestion of food, nor will he be made better or worse by eating improper food. The functions of the large intestine are not necessarily interfered with, but when they are disturbed the patient suffers from constipation and flatulency in addition to the other symptoms.

After a time the general health of the patients is considerably affected. They feel entirely unfitted for business. They may lose flesh and strength to a certain extent, and sometimes this is attended with a certain amount of mental disturbance; the patients become very anxious about themselves, and will imagine that they are suffering from some organic disease. Such mental disturbances are much more apt to occur when there is added functional dyspepsia incidental to disturbances of the liver. Patients, as a rule, have a number of attacks of intestinal dyspepsia, and with each succeeding attack their suffering is prolonged and more severe. Very frequently the first attack will not excite the patient's attention very much. He will feel for a few days that he is not as well as usual, but after that time the pain will pass away and he will pay no particular attention to it. After a time, however, he will have another attack, and finally the attacks become more frequent and severe, and the patient passes into a permanent condition of persistent disturbance of the general health. It is at this time that patients usually seek medical advice.

Treatment.—These patients are not very much benefited by the use of drugs, but they are decidedly benefited by proper hygienic measures. If you can regulate their life, you may do them much good. The best plan for them to adopt is to move from place to place and, if possible, continue traveling for a considerable length of time. If they can afford it, you should advise them to travel for one or two years, and if they can do this, the symptoms will not only disappear, but they will not return, and the patient will thereby be permanently cured. If this plan of treatment cannot be carried out, you should advise the patients to take some out-of-door exercise, such as horse-back riding, but if he cannot do this he should take gymnastic exercise: walking is better than none, but it is not very efficient.

As regards the food of these patients, they should avoid the use of fats and starches, in other words, they should live principally on meats and fruits, and those vegetables that contain the smallest amount of starch. If you give any drugs, those which are the most efficient, as a rule, are ipecac and cubebs in the form of powder. Bismuth, and sometimes iron and quinine, are also given, the last two, however, being administered rather as a tonic than for any immediate effect they have upon the condition of the intestine. If possible, hygienic measures should be first used, and if these fail drugs may then be given.

INFLAMMATORY LESIONS OF THE SMALL INTESTINE.

We meet with certain inflammatory lesions of the small intestine, which vary according to the particular part that is involved. Of the affections of the small intestine we need only give our attention to the inflammatory process which involves its whole length and to that which involves its lower part. We will not here consider inflammation of the duodenum.

ACUTE CATARRHAL INFLAMMATION OF THE SMALL INTESTINE, OR CHOLERA MORBUS.—The small intestine may become the seat of an acute catarrhal inflammation which produces symptoms usually known under the name of *cholera morbus*.

Site.—The inflammation may involve the whole alimentary tract, the stomach, small intestine, and large intestine, or it may be confined to the stomach and small intestine, or to the small intestine alone.

Symptoms.—The symptoms will vary according to the extent of the alimentary tract which is involved. If the whole of it be involved, the patient will suffer from vomiting, purging, and cramps in certain of the voluntary muscles, and with these symptoms there will be prostration which is often very marked indeed. If, however, the stomach is not involved and the disease is confined to the small intestine, there will be no vomiting; there will be simply purging and prostration. Usually there are no cramps of the voluntary muscles when simply the small intestine is involved.

Such an attack most frequently comes on suddenly. Very often patients go to bed in apparent good health and wake up during the night and begin at once to vomit and purge. They frequently become very much prostrated. The length of time these symptoms may continue and their intensity vary with the severity of the inflammatory process. If the inflammation is not severe, the symptoms will be of moderate intensity and will only last from two to six hours. If the inflammation is more severe, the symptoms will last for a still longer time. They may continue for several days or as long as a week. If the inflammatory process is still more severe and the patient is old or enfeebled by previous disease or bad habits, he will pass into a typhoid condition, and may even develop cerebral symptoms, and will finally die in a condition of extreme prostration.

Differential Diagnosis.—There seems to be a predisposition in some persons to this kind of inflammation, and such individuals may suffer from several attacks. It is very important, in these cases, to make out the diagnosis, for, as you will observe, the symptoms are very much the same as those of acute peritonitis and acute inflammation of the vermiform appendix—typhilitis. At the commencement of the attack it may not be possible to differentiate these three conditions. After twenty-four hours, or a day or so, the diagnosis usually becomes more easy. If it is a case of the form of inflammation of the small intestine of which I am now speaking, the febrile movement subsides, the patient is not very sick, but the pain and tenderness on pressure continue. The prostration begins to pass off and then it is clear that the patient is not suffering from acute peritonitis or typhilitis. Sometimes, it is possible to make a diagnosis at the commencement of the disease, owing to the fact that the patients are not from the first as sick as in the other two conditions. Especially, the febrile movement and prostration are not as marked as in either of the other two conditions.

Treatment.—The treatment depends somewhat upon the severity of the symptoms. The milder cases do not require very much treatment. In the first place the bowels should be opened, and the best purgatives are castor oil and calomel in full doses. If you keep the patients quietly in bed and allow them to eat a little ice and feed them with small quantities of milk, together with a little wine or spirits, you will fulfil all the indications. If the symptoms are more severe, opium during the continuance of the fever and the use of stimulants are especially indicated. The patients should be fed upon a fluid diet and be allowed to eat small pieces of ice, for this is usually agreeable to them. The best means of giving opium is by the hypodermic injection of a solution of morphine from time to time. If the prostration is marked, stimulants are to be used, giving either wines or liquors, according to the amount of stimulation required. The indication for stimulation is especially marked when the disease occurs in old persons or in those who are enfeebled by disease or previous bad living. When there is no longer any fever and the only symptom is pain, ipecac may be given in one-fourth grain doses three or four times a day, increasing the dose until the patients take a grain three times a day. The food, at this time, should be composed principally of meat and fruit. The patients should eat mutton or beef finely cut, and any of the fruits which may be in season at the time of the year that the disease exists. By adopting this plan of treatment the patients improve and get well after a few days. This is one of the conditions in which treatment really does seem to be of some avail. I have seen some of these cases in which no treatment was adopted, or at least, none that was intended to do any good, and the symptoms continued for two or three weeks. But, if the cases are treated in the way I have indicated from the outset, the disease will run a much shorter course, and terminate at the end of a week.

Prognosis.—The prognosis of this disease is, as a rule, good. Most of the cases will, indeed, be so severe that you may be considerably alarmed as to the welfare of the patient, but, after a time, they will recover completely. The prognosis is only bad when the disease occurs in old persons and in those who have been imperfectly nourished.

LECTURE XXXV.

DISEASES OF THE INTESTINES—CONTINUED. —MEMBRANOUS ENTERITIS. — DIARRHŒA.

Chronic Catarrhal Inflammation of the Small Intestine.—Membranous or Follicular Enteritis; Pathology, Occurrence, Symptoms, Prognosis, Treatment.—Diarrhœa; Etiology, Symptoms, Treatment.—Diarrhœa Due to Nervous Influences; Symptoms, Treatment.

CHRONIC CATARRHAL INFLAMMATION OF THE SMALL INTESTINE.—*Gentlemen:* Chronic catarrhal inflammation of the small intestine, although common enough, does not give rise to any very marked symptoms. It usually occurs as a secondary lesion to diseases of the heart, lungs, and liver, and is found in connection with Bright's disease and phthisis. Under all these circumstances, this condition of the intestine gives rise to no special symptoms by which we can determine its existence. It does not cause diarrhœa or pain. It is, indeed, possible that it has some effect upon the nutrition of the patient, but there is no special means of ascertaining this. After death from the above-mentioned diseases, there is frequently found a condition of chronic catarrhal inflammation of the small intestine.

MEMBRANOUS OR FOLLICULAR ENTERITIS.—There is still another affection of the intestine which is recognized by its symptoms and not by its lesion. This condition is usually called membranous or follicular enteritis.

Pathology.—It is supposed that the lesion is situated in the large intestine. The disease is not fatal, although it may continue for a considerable length of time. It seems to be a form of *chronic catar-*

rhal inflammation of the colon, characterized by an excessive production of mucus.

Occurrence.—This disease usually occurs in persons during middle life, but it has also been observed in very young children. It is, for some reason, much more frequent in women than in men, especially in women who are suffering from uterine disease.

Symptoms.—The patients who suffer from this disease may have only a single attack, or they may have a number of them. Each attack is characterized, in the first place, by the existence of colicky pain in the abdomen, and this is followed by a diarrhœa of a peculiar character. The patients have large and loose watery passages. In these there is found a certain amount of fecal matter, but not a great deal. In addition, there are found long strings and flakes of coagulated mucus. So firmly is this coagulated that it looks more like coagulated fibrin. When it is first discharged from the bowels, it is of a transparent color, but after being put in water or alcohol, it becomes whiter and denser. It occurs sometimes in the form of flakes; at other times in long strings, cords, or tubes, the tubes looking as if they were formed by the coagulation of mucus upon the inner surface of the intestine, which subsequently becoming detached, come away in the form of tubes. The mucus is a characteristic feature of the disease. A patient may have one, two, or several such movements of the bowels during the twenty-four hours. There will be colicky pains, followed by a large, loose, watery movement. Such an attack lasts only about a week. There will be no febrile movement and no constitutional symptoms. The patient will not even be confined to his bed and will remain up attending to his business. The attack, after lasting for a week, will usually terminate and the patient may never have another. More frequently, however, patients have other attacks, separated by intervals, varying from a few days to several months. If a patient have more than one attack of the disease, his health between the attacks is usually not good. Such patients are likely to suffer from gastric, intestinal, or hepatic dyspepsia, and with these dyspeptic symptoms there will be most commonly some mental disturbance and the patients will become very much alarmed about themselves. They will think that they are losing their understanding, and usually take a very serious view of their case, being impressed with the opinion that they are suffering from some disease which will prove fatal.

Prognosis.—The prognosis is usually good. The patients recover, but only after a long time. It may be three, five, or ten years before they will be completely rid of these attacks.

Treatment.—The disease is one that is not very amenable to treatment. The patients, during the first attack, always seek the advice of a physician and they have been treated in a variety of ways. Sometimes, the disease will appear to be arrested under some particular treatment, but the symptoms will again manifest themselves after some months. The most efficacious treatment is undoubtedly attention to the general health of the patient, and to give such drugs as will correct the dyspeptic symptoms and to insist upon a proper amount of exercise. If possible these patients should have a change of climate; they should travel. If the general condition of the patient can be improved, the attacks will cease and will not return. The two measures which seem to have the most direct effect upon the condition of the intestine are the use of ipecac in moderate doses, one-fifth to one-half a grain in pill form three or four times a day, and the use of cold water (not ice water) enemata. These enemata should be given in as large quantities as the patient can tolerate. Most adults can begin with an injection of half a pint in the morning. As the patients become accustomed to their use, they may be gradually increased up to two pints, which is usually the limit of tolerance.

DIARRHŒA.—We come now to the consideration of diarrhœa, and by this is meant that a patient has during the twenty-four hours a number of loose, partly fluid fecal evacuations. These evacuations may not be accompanied by any pain whatever. They are sometimes, however, preceded by colicky pains, but are not accompanied by them at the moment the evacuation takes place. If there is any pain at all, it precedes the evacuation of the bowels.

Etiology.—Among the causes of diarrhœa are certain conditions of the water, certain nervous influences, and the formation and cutting of teeth in young infants. The disturbance is a functional one, dependent apparently directly upon one of the three above-named conditions. There is no lesion of the intestine. Certain kinds of food produce diarrhœa. There are certain fruits which have, in some individuals, a direct purgative effect. Some people can never eat grapes, melons, or cucumbers, without such indulgence being followed by diarrhœa. These act upon the bowels as would a dose of sulphate of magnesia. Food which is taken into the stomach and passed into the intestine in an undigested condition may act as an irritant and set up a diarrhœa, which will continue as long as the undigested food remains in the intestinal tract. Besides the effect of food in producing diarrhœa, it is well known that water can also give rise to it. There is a great difference in individuals as to their susceptibility in regard to water. There are some persons who cannot drink a single tumbler of cold water before breakfast without its producing immoderate diarrhœa. There are many persons who, when travelling from place to place, find that a change of water produces in them a diarrhœa. In some places the water of wells is contaminated by fecal or vegetable matter, and this again will produce, in those who drink it, diarrhœa of a greater or less severity. When there is inflammation of the upper part of the large intestine, this will produce diarrhœa. In many cases of Bright's disease, diarrhœa is found as a complication, and either with or without chronic catarrhal inflammation of the small intestine. The same is true of phthisis. In a very considerable number of cases, especially toward the close of the disease, there will be diarrhœa. In some cases there will be a lesion of the intestine, but there are other cases in which the diarrhœa exists and is quite severe, and yet, after the death of the patient no intestinal lesions are found. Constipation is another cause of diarrhœa. If the bowels are habitually constipated, in a certain number of patients, this will give rise to a diarrhœa of a peculiar kind. Finally there is a diarrhœa common to children, being distinct from that occurring in adults.

Symptoms.—Diarrhœa, dependent upon nervous influences and upon the condition of the water, is, as a rule, not very severe. The patients will have during the twenty-four hours a number of loose, fecal passages. In a moderate number of cases this condition will cease of its own accord—in the course of twenty-four or forty-eight hours. In other cases, if the patients are left to themselves altogether, as they sometimes are, they may continue for two or three weeks without getting any better, and yet, during all this time, there will be no lesion of the intestine, the diarrhœa being due simply to the condition of the water or the existence of nervous influences.

Treatment.—The treatment in these cases is very simple. A very strict diet is not indicated. Patients may continue to eat ordinary articles of food. One indication is the use of opium, but in moderate doses. It may be given alone or combined with bismuth. Perhaps one of the best forms of opium for this purpose is the ordinary household remedy, paregoric, given to adults in doses from half a teaspoonful to two teaspoonfuls, and to children from five to ten drops. This is usually all that is necessary to be done to check and control the diarrhœa. If it is possible, patients should be required to keep themselves very quiet, especially during the hot weather, and it is during hot weather that the disease occurs most frequently.

Diarrhœa Due to Nervous Influences.—There is another form of diarrhœa occurring in adults, and dependent, principally, upon nervous influences, which merits separate mention. This form of diarrhœa appears to have no particular relation to the weather. It occurs as frequently in cold as in warm weather, but seems to depend upon those conditions which we are accustomed to group roughly under the name of nervous influences. This form of diarrhœa belongs peculiarly to adults. It does not occur, so far as I know, in children. I have not seen any examples of it in very old persons. It occurs especially among the better classes, among persons engaged in business, or in those belonging to the different professions. It does not belong to the laboring classes. Not infrequently it occurs in persons who are overworked, but whether it is dependent upon overwork or not we cannot say ; certain it is that a number of cases give the history of overwork of the patient before the diarrhœa begins.

Symptoms.—The diarrhœa commonly begins in the morning. The patient will find after having had an ordinary natural movement of the bowels in the morning that within the course of two or three hours he will have one, two, or three large, loose watery movements, and the desire to have these will be very sudden and irresistible, the patient being unable to control himself. The disease, in a certain number of cases, will not get any worse than this. Every morning, or almost every morning, the patient will have one, two, or three of these large, watery movements. If the patient is properly treated at this time, he may recover and never have another attack. In other cases, the patient does not get well and the symptoms become worse. Now, instead of having only these movements in the morning, he will have them in different parts of the day, in the morning, afternoon, and evening, and, as a rule, very soon after taking food. After the disease has lasted for some time, the relation between the taking of food and the diarrhœa becomes very marked. Very soon after beginning to eat food, the patient will suddenly be taken with an irresistible desire to evacuate his bowels, and will be obliged to leave the table for this purpose, after which he may return and finish his meal. In this condition these patients may continue for several years. They will go on in this way for two, three, or four years, and during the greater part of the time will suffer from this condition of the bowels, every day having from two to six large movements which come on very soon after the ingestion of food. When the disease is protracted in this way the patients become emaciated, but not to a very great extent. They cannot attend to business and are apt to become anxious about themselves. They may become nervous and watch the condition of the bowels, counting every day how many movements they have. They may make themselves worse in this way by constantly worrying about the condition of their bowels.

Treatment.—In the milder cases of the disease and in which the duration has not been prolonged, the treatment of these patients is tolerably simple. The patients are quite amenable to treatment. The drugs which are most serviceable are one of the preparations of opium (codia), ipecac, bicarbonate of soda, and hypersulphate of soda. In some cases you may combine these drugs. A prescription which answers very well, is the following :

Codia	gr. $\frac{1}{6}$.
Pulv. ipecac	gr. $\frac{1}{5}$.
Sodæ bicarb.	gr. iij.

in pill form, to be taken after each meal.

Besides these slighter cases, we meet with others in which the disease becomes of more consequence. The loose movements occur, not only in the morning, but in the middle of the day and in the evening. The ingestion of food has a marked effect in exciting a disposition to these loose fecal movements, so that, as soon as the patients begin to take food, they experience a desire to have one of these loose, watery movements. The disease may extend over a period of two, three, or four years. It is not, indeed, always continuous throughout the whole of this time. There may be periods of weeks or months during which the patients seem to be better, after which the symptoms make their appearance as before.

The ordinary drugs which are used in the treatment of diarrhœa, such as preparations of opium, ipecac, or the various astringents, are of no service whatever in the treatment of this condition. It is necessary to treat these patients in a different way. The measures which should be adopted are, in the first place, hygienic. The diet should be regulated. It is not necessary to starve the patient or deprive him of any of the regular articles of food ; but each of the three meals should be composed of food which is easily digested. Exercise does not seem to particularly benefit this form of diarrhœa. If drugs are used they should be of the class of tonics, such as iron, quinine, and the mineral acids. One of the best tonic drugs is the citrate of iron and quinine given in solution. A measure which is of very great efficacy is the use of cold water enemata every morning after breakfast, in the same manner as recommended in the treatment of membranous enteritis, beginning with half a pint of cold water and gradually increasing until the point of tolerance is reached. The water should be thrown into the rectum very slowly. When the extreme limit of the capacity of the intestine is reached, the water and fecal matter will be expelled. In addition, these patients are often benefited by taking a tumblerful of ordinary effervescing Vichy water either alone or with a teaspoonful of whisky or brandy added. If this treatment is thoroughly carried out, its good effects are manifest and lasting. In this way it is possible to cure a patient of diarrhœa which may have already lasted two, three, or four years. Of course, in the treatment of these cases, there is always need of a certain amount of management and detail, which it is impossible for me to give, but the above are the general principles which should guide you.

LECTURE XXXVI.

DISEASES OF THE INTESTINES—CONTINUED. —DIARRHŒA IN ADULTS.

Diarrhœa caused by Food; Occurrence, Symptoms, Treatment.—Diarrhœa Complicating Bright's Disease; Treatment.—Diarrhœa Complicating Pulmonary Phthisis; Pathology, Treatment.—Diarrhœa Complicating the Cure of the Opium Habit; Treatment.—Diarrhœa Dependent upon Constipation; Treatment.

DIARRHŒA CAUSED BY FOOD.—*Gentlemen:* I have already told you that food may produce diarrhœa in one of two ways. Either the articles of food are of such a character as to act directly as purgatives, as is the case with some of the fruits, or the food taken into the stomach passes into the intestine undigested, and remaining there, acts as an irritant.

This sort of diarrhœa is seen much more frequently in children than in adults. There are many of the ordinary articles of diet which, from time to time, children, for some reason, do not seem to be able to digest, and under such circumstances a temporary diarrhœa is set up.

Symptoms.—Such a diarrhœa is usually attended with more or less colicky pains, in addition to a number of loose fecal movements. In some cases the article of food which has given rise to the diarrhœa will pass through the intestinal canal within a few hours, and when this happens the diarrhœa ceases without treatment. In other cases it is not gotten rid of so quickly, and the diarrhœa may continue for several days.

Treatment.—The first indication in the treatment of these cases is evidently to remove from the bowels the substances which are irritating them. This is the class of cases in which the use of purgatives is specially indicated. It is not wise to give at first opium or any sedative medicine, but a purgative should be given. Castor oil, rhubarb, or one of the saline cathartics, or any cathartic which acts pretty promptly, will answer. In many cases this is all that is necessary, for after the cathartic has acted upon the bowels and the offending substance is removed, the diarrhœa ceases. Sometimes, even after this, the diarrhœa continues, and then the indications are to use sedative medicines, such as one of the preparations of opium given by itself, or in conjunction with bismuth or bi-carbonate of soda.

Diarrhœa Complicating Bright's Disease.—The diarrhœa which occurs as a complication of Bright's disease is sometimes, apparently, merely an effort on the part of nature to get rid of the dropsy. Patients suffering from Bright's disease accompanied with dropsy and the passage of a greatly diminished quantity of urine, are very apt to have diarrhœa. This requires no treatment, the patients being better on account of it.

In other cases of Bright's disease the diarrhœa continues although the patients are passing a considerable amount of urine. It may be so profuse as to hasten very much the death of the patient. This form of diarrhœa, occurring as a complication of Bright's disease, is, perhaps, the most intractable of any which we are called upon to treat. It is not considered safe to give opium in large doses, and the small amount which it is safe to give is of no use in controlling the diarrhœa. The same is true of ordinary astringents, alkalies and mineral acids, none of them being of any efficacy in checking the diarrhœa. In fact, I am quite unable to tell you of anything that will check it.

Diarrhœa Complicating Pulmonary Phthisis.—The diarrhœa which occurs in connection with pulmonary phthisis is, however, much more amenable to treatment. It is always difficult to be sure of the exact character of the diarrhœa which complicates phthisis. In some cases it depends upon ulceration of the lower part of the small intestine; in others, it is dependent upon a chronic catarrhal inflammation of the lower end of the small intestine and colon. Again, it exists without any lesion whatever of the small intestine or colon, and it is very difficult to say to which of the above classes the diarrhœa belongs.

The treatment is usually the same, no matter what the condition of the intestine may be. It is customary to give opium by itself, or combined with bismuth, or one of the mineral acids, which plan will answer very well to check the diarrhœa.

Diarrhœa Complicating the Cure of the Opium Habit.—In patients who have been the subjects of the opium habit and have ceased to use the drug, it is common enough for them to be troubled with the diarrhœa. If these patients try to cure themselves, or to be cured of the habit of the constant use of opium, the bowels are very apt indeed to become loose, and the diarrhœa continue for a considerable length of time. It may distress and enfeeble the patient to a considerable extent.

A certain number of cases of this form of diarrhœa may be controlled and permanently cured by the use of ipecac, given in the form of pills, in doses of one-fifth of a grain to a grain, according to the tolerance of the patient. Unfortunately, however, such good results do not follow the treatment of all cases. Where it fails, the mineral acids, dilute muriatic acid, sulphuric or nitro-muriatic acid, may be used. Opium should not be administered, for, by giving it, even in small amounts, the work of having to break off the habit will have to be done over again.

Diarrhœa Dependent upon Constipation.—There is still another form of diarrhœa which is dependent upon constipation—upon the accumulation of fæces in the colon. There is one form of diarrhœa of this kind which we see quite frequently in old persons, usually in those over seventy years of age, or it may occur in persons a little younger than this. In such individuals there is very apt to be constipation, the sluggishness of the bowels seeming to depend principally upon the weakness of the muscular coat of the colon. In consequence of this, there is an accumulation of fæces in the rectum. During the accumulation of this mass of fæces constipation has not been absolute. There have been imperfect and infrequent movements of the bowels. When the accumulation has reached a certain point the patients begin to have diarrhœa. During the twenty-four hours they will have a number of loose fecal passages, which come, of course, from above the point where impaction exists, so that the patient suffers, at the same time, from constipation and diarrhœa. There is an accumulation of fæces in the lower part of the rectum which is not discharged, and in addition, there are loose fecal passages.

In some patients, this condition can be relieved by an ordinary cathartic, such as castor oil, aloes, or rhubarb. By these means the mass of accumulated fæces may be discharged completely or partly, and the patient will be relieved for a time. In other cases, this treatment will not be sufficient. It may simply increase the diarrhœa, the hardened fæces remaining in the lower part of the rectum. When this happens, it is necessary to dislodge the fæces by mechanical means. With the finger, or a spoon, or an instrument of some kind, you have to dig this mass out of the rectum. It is a very dirty and disagreeable business, but it is the only way in which these patients can be relieved. Sometimes even this plan is not altogether satisfactory, on account of the fæces extending up so high. Under such circumstances you have to resort to the use of enemata. For this purpose you use a so-called fountain-syringe, in which the water is propelled by the force of gravity, and use it for the purpose of irrigating the rectum rather than giving an enema. After a certain amount of water has run into the rectum it begins to run out again. The irrigation is continued for the purpose of softening and washing away as much as possible of the hardened fæces. In one or other of these ways it is possible, with more or less difficulty—often with a good deal of difficulty—to succeed in clearing out the lower part of the rectum. When this has been accomplished, the patient will soon be in a normal condition. Care should be taken to prevent the reoccurrence of a similar condition, and for this purpose the patient should be placed upon the use of some drugs which will act in such a way that the large intestine will be emptied every twenty-four hours. The best drugs for the accomplishment of this are strychnine, one-fortieth of a grain, and aloes, one grain, in pill form, one to three times a day, as is necessary to have the desired effect upon the bowels. It is exceedingly important that this condition of the bowels should be recognized and relieved. The fact that after the fæces have accumulated to a certain point the diarrhœa sets in, often leads to the non-recognition of these cases. An old person, in regard to whose bowels you have no previous knowledge, will come to you with the story that he has a diarrhœa. It is very easy to think that the diarrhœa is of the ordinary kind, and hence be led to give a sedative medicine, which, of course, will make the patient worse rather than better. It is very important to recognize this, for it is a condition which is capable of producing death. Every year there occur deaths simply from the result of an accumulation of fæces in the lower part of the large intestine, which has not been relieved. If this condition is not relieved after a long time, the patients become very much prostrated, and die in a condition resembling collapse ; the pulse becomes feeble, there is no febrile movement, and they finally die exhausted.

Besides diarrhœa dependent upon constipation of this kind, there is a diarrhœa dependent upon constipation of a different character. The second form is not nearly as common as the first. The first is very common indeed, and you should always be on the lookout for such cases. You should never treat a diarrhœa in a person over seventy years of age without thinking of the possibility of the existence of fecal impaction. In this second set of cases, the disease or condition is not confined to old persons. It occurs in adults as well as in others. There is not any fecal impaction, properly speaking ; there is no place in which there are hard, dry fæces. But, instead of this, as is found after death, the whole length of the intestine is filled with soft tenacious fæces, and may even be distended to a considerable extent. It is very difficult, indeed, to understand how this accumulation of fæces takes place. There is no mechanical obstruction of any kind, and yet, for some reason, the fæces accumulate in this way. These cases do not usually come under observation until a few days before death. My personal knowledge of them is confined to hospital cases, which have not been seen until a few days before death. These patients come into the hospital complaining of diarrhœa. They tell you that for some days they have been having a number of loose fecal passages. The diarrhœa continues, but you notice that the patients appear to be more prostrated than in ordinary diarrhœa, and there is a certain distention of the abdomen. There is no febrile movement, as with cases of constipation in old persons ; these are very apt to pass without the true condition being recognized. Patients are given opium, bismuth, and mineral acids in the ordinary way, the diarrhœa is checked, and in a few days the patient dies, and when you make the post mortem examination you find the large intestine filled with soft fæces. Here, again, the important point is to recognize the actual condition. Instead of trying to control the diarrhœa by opium, the indication, of course, is to give purgatives, and endeavor to empty the large intestine.

LECTURE XXXVII.

DISEASES OF THE INTESTINE—CONTINUED.—DIARRHŒA IN CHILDREN.

Diarrhœa in Children; Symptoms, Treatment.—Dysentery in Children.—Other Cases of Diarrhœa; Treatment.—Cholera Infantum; Pathology, Occurrence, Symptoms, Duration, Prognosis, Treatment.

DIARRHŒA IN CHILDREN.—*Gentlemen:* We come now to the consideration of diarrhœa in children. Diarrhœa is, as you know, one of the most frequent morbid conditions of early childhood, especially of children under two years of age, or during the period of dentition. There are comparatively few children in our climate who reach the age of two years who have not suffered from some form of diarrhœa.

Symptoms.—In these young children we meet with a very mild form of diarrhœa. The child will be apparently a little sick. There will be no febrile movement, but there will be loss of appetite, and the child will be less disposed to play, and more irritable than normal. During the twenty-four hours there will be a number of loose fecal passages, which are, as a rule, of a greenish color. The disease occurs most frequently in hot weather, especially during the hot periods of several days duration which we have every summer. The mild cases may get well without any treatment whatever. In other cases, although the diarrhœa is of a mild form, there is a disposition on the part of the patient to continue to have, day after day, a number of loose greenish passages. The indication for treatment is the use of sedatives, and of these the most reliable is opium. In young children, however, the dose of opium which is given should be small and in the form of Dover's powder or paregoric. It is usually best to give it in combination, as with the subnitrate of bismuth, or small doses of ipecac, rhubarb, or bicarbonate of soda. In addition, these children should be kept quiet and in a cool place. Their diet should be composed principally of milk and some of the starches, either rice, arrowroot, or bread.

In other cases, the constitutional symptoms are more marked, although the diarrhœa is about the same. The stomach is very often disturbed, and, besides the diarrhœa, there will be vomiting, which is not commonly very persistent. The patients will vomit from time to time, especially if they take too much food of any kind into the stomach. There will be a febrile movement, and this in young children will be quite marked. A very little thing will be sufficient in a child under two years of age to give rise to a temperature of 102,° and not infrequently there may be a temperature of 103° or 104°. The febrile movement is sometimes continuous, sometimes intermittent. When intermittent, it is sometimes quite difficult to say whether the child is suffering from this disease or ordinary malarial poisoning.

Treatment.—In these cases, although the constitutional symptoms are more severe, the indications for treatment are just the same. The patients should be kept quiet. In some cases, it will be necessary to begin with a purgative. Such treatment is indicated when there is a history of the child having eaten some improper food and having been affected by the weather. To other patients, where there is no such history, sedatives are given at once, and here, as in the other cases, opium may be given alone or in combination with rhubarb, ipecac, or soda. There are some of these cases, however, in which these remedies are not sufficient to control the disease entirely. The child gets somewhat better, but it does not get completely well. There is still left some looseness of the bowels and some prostration. The child remains irritable and peevish, and has a number of loose passages during the twenty-four hours. In such cases, there is no use of persisting in the administration of opium. The best possible plan of treatment to adopt is to send the child to another place, where the air is different from the one in which it lives. If the child is taken sick at the sea-shore it should be sent inland. If it is taken sick in the country it should be sent to the city. If taken sick in the city, it should be sent to the sea-shore or to the country. It does not make so much difference where the child is sent, only provided it has a change of air and surroundings. This alone will put an end to the diarrhœa in a considerable number of patients. There are many cases in which such a plan of treatment cannot be carried out, and, under such circumstances, other drugs than those mentioned have to be tried. Perhaps the best for this purpose are the preparations of mercury, either calomel, or mercury with chalk (hydrargyrum cum creta). At first, a small dose should be given, half a grain two or three times a day. During its administration the child's passages should be watched in order to ascertain if they become more natural in consistency. When the passages have reached the standard of health, its administration may be discontinued altogether. In those children in which the disease is somewhat protracted, it is always necessary to pay particular attention to the food. If the child is nursing and if the nurse or mother is in good condition, it should be allowed to continue to nurse. If, however, the child is being fed with a bottle, it will usually be necessary to change the food. Most children fed with a bottle are fed upon ordinary milk and water, or condensed milk and water, or with milk and some form of starch, either Graham flour, ordinary flour, or rice flour. When the child is taken sick, you will usually do well to change the food. If the child has been taking condensed milk and water, give it milk with starch or rice flour, or it may be fed upon cream and water for a few days. If the child is somewhat older than this and is no longer nursing, good results will be obtained by feeding it upon uncooked beef. A piece of beefsteak should be taken and scraped with a knife so as to make a fine pulp of the meat, to this can be added a little salt, and upon it the child can be fed for a considerable length or time. It is possible to feed children upon this raw meat a very long time, but, in doing so there is danger of producing scurvy. I have seen an instance of this in the case of a child who, when he had diarrhœa, was placed upon a meat diet, and after recovering from the disease the parents continued to feed him in the same way for a year, at the end of which time the child developed well marked symptoms of scurvy.

Dysentery in Children.—There are other cases of diarrhœa in children in which the child has a few loose fecal movements which finally stop and the child passes small amounts of blood. These are really cases of dysentery rather than diarrhœa, but it is convenient to speak of them at the present time. In these cases of dysentery, it is well to begin with a dose of castor oil, especially if you see the patient at the beginning of the attack. You give a teaspoonful, or two or three teaspoonfuls according to the age of the child, together with a little laudanum. In some cases, after the bowels have been acted upon in this way, there will be no further dysenteric symptoms. When, however, these symptoms continue, the use of opium is indicated and is best given in small doses combined with bismuth.

Other Cases of Diarrhœa.—There are still other cases in which the disease begins as a mild diarrhœa, or it begins as a dysentery. The child gets no better, notwithstanding the use of drugs and the regulation of the diet. If it begins as a dysentery the dysentery ceases and is succeeded by a diarrhœa. If it begins as a diarrhœa, the diarrhœa continues, but the character of the passages changes. The passages, instead of being green, now become white. They are soft, not fluid, and of about the consistency of starch. They become whiter and whiter, until almost of a dead, opaque white color. There is not only a change in the color of the stools, but there is an increase in the quantity. The child will not have a great many movements during the twenty-four hours, but each of the movements is very large—sufficient to fill a chamber at each evacuation. The passage of such large and peculiar stools is something very strange. During the progress of the disease the child emaciates very rapidly; the whole body becomes thin and weak; there is a drawn and anxious expression of the face and the mouth becomes dry. During the process of emaciation the abdomen remains large and distended. The appetite of these children, as a rule, is abnormally increased. They are constantly thirsty and hungry. They remind you in this respect of a patient suffering from diabetes. They will drink and eat nearly all the time, if allowed to do so. The large white movements continue, the child gets worse and worse, and, if the disease is unchecked, it finally proves fatal, the child dying exhausted.

Treatment.—If, however, the disease has not lasted for a long time, it is quite amenable to treatment, and when this is properly directed, the patients will recover, at first slowly, then more rapidly, and finally get well altogether.

The drugs which seem to be of most decided benefit in these cases, are the mineral acids, especially dilute hydrochloric acid, given in doses of from ten to twenty drops three or four times a day, according to the age of the child. At first, there should be added to each dose of dilute hydrochloric acid a small amount of opium, one, two, or three drops of the tincture of opium. As the child improves, the quantity of opium is diminished, and after a time discontinued altogether. The administration of hydrochloric acid is continued until some time after the child seems entirely well. It is best to give these children milk, cod-liver oil, or meat, according to their age. In other words, it is best to feed them upon fat and meat, giving them as little starchy food of any kind as possible. If they are very young, they do best upon cream and water. Older children are best fed upon cream and milk mixed, and to this may be added, meat and cod-liver oil. In these, as in all cases of diarrhœa, a change of climate is always of very great benefit. If it is possible to move a child to an entirely different region, the improvement will be much more rapid; but in these chronic cases a change of climate is not sufficient: some medication is required as well.

CHOLERA INFANTUM.—The severest form of diarrhœa in children is usually called by the name of *cholera infantum.*

Pathology.—This form of diarrhœa proves fatal in many cases, so that there is an opportunity offered for ascertaining whether there are any characteristic lesions of the intestine to account for the disease. Although this opportunity is afforded us, as a rule no marked lesions are found, notwithstanding the fact that the symptoms are very severe during life. The change in the intestine consists, principally, of the swelling of the agminated glands of the small intestine and the solitary glands of the large intestine. In the most marked cases the swelling will be succeeded by a softening of the glands, and they will become broken down. In this way ulcers are formed. There is, in these cases, a swelling of the lymphatic glands. In some cases there will be no lesion in the intestinal tract. The intestine will not even be congested; sometimes it is pale. Ordinary fæces may be found in the intestine, but more frequently they are composed of a thin, whitish fluid, resembling somewhat the rice-water fluid found in the intestines of patients who die from Asiatic cholera. This fluid is often of a more brownish color.

Occurrence.—This, like other forms of diarrhœa, occurs in very young children; most frequently in those under two years of age. It affects children most frequently who are artificially fed. It occurs among children very frequently who are crowded together, as in foundling asylums, or in places where a considerable number of them are fed artificially. Under such circumstances the mortality is very great indeed. A number of years ago, when a great many children were reared on Randall's Island, nearly all that were then there died during the summer months from this disease. In the same way, but to a less degree, the mortality is increased in tenement houses where the poor are crowded together in large numbers. Every year deaths from this disease constitute the larger part of mortality in New York.

Symptoms.—The symptoms of this disease follow sometimes ordinary simple diarrhœa; sometimes they come on suddenly. The child will have a diarrhœa for some days or weeks, and then the symptoms of cholera infantum will come on. In other cases, the child will have been in perfect health, when the symptoms may make their appearance suddenly. The most marked of these symptoms are vomiting and purging. The vomiting is not as constant as the purging; it is not always present. It may continue during the entire course of the disease. At first, a child simply vomits whatever food there is in the stomach, but, after vomiting this, it continues to vomit a thin, watery fluid, which is sometimes of a brownish color. Not only this, but everything which is taken into the stomach is rejected. The severity and continuance of the vomiting varies. In some cases there may be no vomiting; in other cases, vomiting only at the commencement of the disease, or there may be incessant vomiting.

The purging is always present. The children do not have merely ordinary diarrhœa, but there is real purging. The movements from the bowels are very frequent. A child may have a movement from the bowels every ten minutes for twenty-four hours, or even longer. At first the matters which are passed are simply fluid fæces. After this the child passes thin watery fluid, which is sometimes of a brownish or dirty white color; but it is not usually passed in great quantities. With the purging there may be a certain amount of colicky pain in the abdomen come on before the movement. This pain, however, is not always present. Commonly there is no febrile movement at first, but occasionally there is. The prostration is very marked indeed, and comes on very early. Within a few hours after the commencement of the symptoms, the whole appearance of the child will be altered. the pulse will be feeble, the eyes will have become sunken, the face will have a haggard, fixed, and anxious expression, and the skin will be cold. After the disease has lasted longer, these symptoms become more marked and there is added rapid emaciation. A well-nourished child in three or four days will be reduced almost to a skeleton. There is scarcely any disease in which emaciation occurs so rapidly as in cholera infantum. The urine in many cases is diminished in quantity, and may be very scanty or almost suppressed. In the bad cases cerebral symptoms are developed. The child becomes very restless, does not sleep, or sleeplessness may alternate with unnatural drowsiness, and this may pass fairly into stupor or even into a condition of semi-coma. There are twitchings of the voluntary muscles, and there may even be general convulsions. It may look, in some respects, like one suffering from tubercular meningitis or acute hydrocephalus. If the disease is to prove fatal, the purging and prostration become more marked, the cerebral symptoms more pronounced; and, finally, the child dies in a state of collapse, with an imperceptible pulse, cold, clammy, livid skin, or dies in an attaek of convulsions or in a state of coma.

Duration.—The duration of the disease varies with its severity and the previous condition of the child. The more severe cases occur in children who have been previously badly nourished. In such, the disease may run its course and prove fatal within twenty-four hours. More frequently, however, the disease continues for a number of days, and often as long as a week. At the end of this time the disease either proves fatal or the child gradually gets better. Occasionally there will be a remission in the symptoms for from one to three days. The child will apparently be better, and considered likely to recover. But this temporary improvement is succeeded by the return of the symptoms, and the child may finally succumb to the disease after a somewhat protracted period—sometimes as long as two weeks.

Prognosis.—The prognosis of this form of diarrhœa is always very serious, a very considerable number of the cases proving fatal. It is more likely to prove fatal in young infants than in children somewhat older. Children brought up on the bottle are more likely to die of the disease than those who have been nursed. Badly nourished children are more apt to die than those whose health was good previous to the attack. But there are no cases of cholera infantum which do not cause a certain amount of alarm. Even children who have been in the best of health die not infrequently from the disease.

Treatment.—The treatment of this disease, it must be confessed, is not very satisfactory. The disease is not controlled by the treatment, but runs its natural course. You may help the disease, but no remedy, so far as I know, has any decided effect upon it. If it is possible, these patients should be sent to a different place. This is, as I have said, good in all cases of diarrhœa in children, but it is especially so in the case of children having cholera infantum. The earlier the child can be moved the better for the health, and you need never hesitate to have the child moved no matter how sick it may be. Even if the prostration is so extreme as to indicate that the child will die in a few hours, you are still justified in sending it away from the place where it was taken sick, for, after reaching a different climate, such a child may rally and get better. You should never wait for the child to get strong or better before it is sent away, but it should be taken away as soon as possible. With infants that are nursing it is, as a rule, best for them to continue to do so. If the child is bottle-fed, I think it will usually be found of advantage to feed it upon cream and water, or upon wine whey. Wine whey is made by boiling a certain amount of milk and adding to it, just as it commences to boil, a moderate amount of sherry wine. In this way the casein of the milk is coagulated and thrown down, and there is left in solution the water and fat of the milk as well the wine which has been used to coagulate the casein. Instead of coagulating the milk in this way, you may coagulate it with dilute hydrochloric acid, after which the milk may be given in the same way in small quantities pretty frequently. If the vomiting is marked, a considerable amount of this will be brought up, but some will remain in the stomach. If the vomiting is very persistent and the child has emaciated considerably, sometimes good results will be obtained by feeding the patients through the skin. For this purpose the skin should be washed thoroughly with soap and water, after which olive or cod-liver oil should be applied and rubbed in very well. The skin will absorb a large amount of fat, and sometimes you will be able to nourish the child in this way which cannot be nourished any other way.

There have been a great many drugs used for the cure of this disorder. Opium, in all its different varieties is employed very largely, but it does not seem to have any very decided effect in checking the action of the bowels. It has a good effect in allaying the restlessness of the children and making them more comfortable. For this reason I think opium in small quantities may be given, but it does not have much effect in checking the purging or altering the course of the disease. Hydrargyrum cum creta, in doses of from one-fourth of a grain to three grains, may be given from two to four times a day, according to the age of the patient. In a certain number of cases the preparations of mercury seem to be of some service. The movements become less frequent and are of a more fecal character. The child may recover from the disease. Some physicians prefer to use the bichloride of mercury or corrosive sublimate. This, of course, is given in small doses from $\frac{1}{100}$ to $\frac{1}{150}$ of a grain three or four times a day, according to the age of the patient. This drug again seems to be of service in some cases. Cod-liver oil is also given in this disease in small and repeated doses; it is never given in large doses, so as to act as a purgative. A few drops made into an emulsion are given every three or four hours. Acids are also given. Dilute nitromuriatic acid, sulphuric acid, and hydrochloric acid are given. The preparations of bismuth are also used. These are the drugs most frequently given, but there are many others which have been employed from time to time. The treatment of a case of cholera infantum should always be begun with a good deal of doubt as to the final results. You do the best you can, but in spite of this I think you will feel that the natural course of the disease is the main point in the case. If it does not run a very severe course, the patient will get well. If the disease runs a very severe course, the patient may die in spite of all treatment.

LECTURE XXXVIII.

DISEASES OF THE INTESTINES—CONTINUED. —CATARRHAL AND CROUPOUS DYSENTERY.

Dysentery; Occurrence, Pathology.—Catarrhal Dysentery; Pathology, Symptoms, Treatment.—Croupous Inflammation of the Large Intestine; Pathology, Local and General Symptoms, Symptoms when Lesion is Confined to Upper Part of Colon, Prognosis, Treatment.

DYSENTERY.—*Gentlemen:* We take up to-day the study of *dysentery.* The word dysentery is one which is used somewhat loosely, and not always with exactly the same meaning. It is sometimes used to designate the fact that a patient has had during twenty-four hours a number of passages composed, not of fecal matter, but of mucous and blood. By others the word is used to designate a definite disease, not a symptom; it is used to designate the inflammations of the large intestine as distinguished from inflammations of the small intestine; it is used synonymously with colitis, and it matters not whether such inflammations of the large intestine are accompanied with characteristic dysenteric passages. In addition, the word is used somewhat indistinctly to designate sometimes the one, sometimes the other of these two conditions, and yet, after all, most of us, in speaking of dysentery, have in mind a definite idea of the particular disease of which we are speaking. We usually mean that there is an idiopathic or primary inflammation of the large intestine, affecting the lower part of the colon rather than the upper, and we expect, as a result of such an inflammatory process, the production of characteristic dysenteric stools composed of blood and mucus. This is the sense in which I use the word dysentery and the condition which I will now describe.

This disease is characterized sometimes as sporadic, sometimes as endemic, and again as epidemic; in a certain number of cases it becomes chronic.

Occurrence.—Sporadic cases of dysentery may occur in almost any climate and under almost any circumstances, but they are much more apt to occur in the fall of the year than at any other season—in the latter part of August, September, and October in our climate. There are some places where the disease is fairly endemic. Such places are in tropical regions and in localities where there are marked malarial influences; where chills and fever and remittent fever are present. In such localities the disease prevails more or less all the time, but may be more prevalent at some particular time than at other times. Epidemics of the disease are not as common now as formerly. During the Middle Ages in Europe every few years there would be a severe epidemic of dysentery, usually in one or other of the larger cities, and the disease would, as a rule, be of a particularly fatal character. Though such epidemics are now no longer common, however, from time to time epidemics do occur. Whenever large bodies of men are congregated together, especially as in armies, dysentery prevails to a considerable extent. It is one of the regular diseases to which armies are subject and may affect large numbers of men. When it prevails in this way, it can hardly be called an epidemic, for it seems to be due rather to the way in which the men live than to any special epidemical influence.

It is still doubtful whether or not the disease can be communicated from one person to another. If it can be so communicated, it is probably accomplished through the fæces. As long as this point is unsettled, we should keep ourselves upon the safe side. It should always be seen to that the fæces of patients suffering from dysentery are not thrown into the privy or watercloset. They should be kept separately and mixed with one of the strong mineral acids (sulphate of iron) which will decompose them, and, if possible, they should be buried in some place by themselves. In some cases the disease seems to be due to the water which the patients drink; and it is sometimes possible to determine that such water is contaminated in a certain way. Occasionally it is found that people who drink water from a certain well have dysentery, and others living in the same locality, and who do not drink the water of this particular well, are free from the disease; but still we cannot find out what changes have taken place in the water. In some instances, however, it is certain that the cause of the disease is to be attributed to the admixture of organic matter with drinking water. In summer resorts the wells and privies are apt to be quite near each other, and when these places become too popular, there will usually come a season when dysentery will prevail to a considerable extent. In these cases it seems to be due to the percolation of fecal matter through the soil into the well and the subsequent contamination of the well-water in this way. The cases of dysentery due to contaminated well-water really make up the larger number of those which are met with in the country about New York.

Pathology.—The lesions of dysentery are inflammatory. The pathological process is an acute inflammation of the large intestine, but the character or type of the inflammation differs in different cases. In some patients the inflammation is of a catarrhal variety; it is simply an acute catarrhal inflammation of the mucous membrane of the large intestine; or it may be an inflammation of the croupous type affecting the mucous membrane of the large intestine; or the inflammatory lesion is situated in the solitary follicles of the large intestine, and in addition the mucous membrane may also be inflamed to a certain extent. The part of the colon which is usually inflamed is the lower end of the rectum. Sometimes only a small part of the rectum will be inflamed in this way. In more severe cases the larger part of the colon will be inflamed, there being two or three feet of it involved, or the whole length of the gut may be implicated. In still other cases, the lower part of the colon will not be inflamed, but the inflammatory process will be confined to the upper part. In the most severe cases of the disease there will be a very marked congestion of the intestine. The entire portion which is involved will be congested to a very unusual degree, and in addition the blood will become stagnant and accumulate in the vessels. The walls of the intestine are very much swollen, of a dark, livid color, and in the worst cases become gangrenous, and thus death may ensue as the result of excessive congestion.

Catarrhal Dysentery.—The catarrhal form of dysentery is that in which the lesion is the result of a simple catarrhal inflammation of the mucous membrane. In this form the lesion is almost always confined to the rectum. It does not involve a very large part of the colon.

Symptoms.—The symptoms of this condition are principally local. A patient who has previously been in good health, will begin to have an ordinary diarrhœa. After this has lasted a day or so—sometimes for three or four days—the fecal passages will cease, and the patient will begin to pass from the bowels small quantities of mucus mixed with blood. The passages of mucus and blood are attended with a considerable amount of pain, which lasts for a little time after defecation. The patient suffers from a feeling of irritability about the rectum during most of the time, and he feels as if he would like to have a passage from the bowels, but when he attempts to do so, he will be able to pass only a small amount of mucus, with or without blood. In other cases there will be no preceding stage of diarrhœa. The patient will, from the first, have characteristic dysenteric passages, accompanied with characteristic pain and irritability about the rectum. In the milder cases there are no constitutional disturbances, no febrile movement, and very little prostration. A considerable number of patients will continue about their daily work, not feeling sick enough to go to bed, or even to remain in the house. Others will have a moderate febrile movement, a certain amount of prostration, and will remain in the house or even in bed. The amount of febrile movement and prostration seem to depend upon the condition of the patient; if young and feeble they will be present, but if the patient is strong, they will not be present.

Treatment.—These cases last for a few days or a week, and then the patient gets well. It is probable that all patients thus affected would get well without any treatment whatever except the keeping of them quiet. The inflammation seems to run a definite course, but, although the disease has this natural tendency, it should be remembered that the patients may suffer a good deal, and hence it is proper to treat them. They should be treated not so much to effect their convalescence as to keep them comfortable. If a patient is seen at the commencement of the attack, the best thing is to give from a dessertspoonful to two tablespoonfuls of castor oil, together with from five to twenty-five drops of the tincture of opium. In addition, perfect rest should be insisted upon. The patients ought not only to remain in the house, but they should remain in bed. If they will not do this, they may lie upon the sofa; it is very important that they should not move about at all. In the milder cases, it may not be necessary to give anything further than I have already indicated. The bowels will thus be freely moved, and although the patient may continue to pass a little mucus and blood, yet the pain and irritability will disappear, and no further treatment will be required. When the pain and irritability continue, opium in some form will be required, and it may be given by the mouth or by the rectum, either in the form of suppositories or small enemata, or it may be given hypodermically. It does not make much difference how it is given, the indication being simply to give enough to keep the patient comfortable. The diet of these patients requires to be regulated. They should not eat solid food. Usually the best food is milk, or milk with one of the preparations of starch, as arrow-root, Graham, or farina. With this treatment the disease runs its course in a few days and patients get entirely well.

From time to time, however, we see cases of this form of dysentery, especially among the laboring classes, in which the patients are not treated at all during the active stage, and not only this, but they even continue about their work. The result under such circumstances is not as favorable as in other cases. The acute symptoms do indeed cease after a time, but the mucous membrane does not return to a healthy condition; and the acute catarrhal inflammation is succeeded by chronic catarrhal inflammation, which may last for months or years. Examples of this are met with quite frequently in hospital and dispensary practice.

CROUPOUS INFLAMMATION OF THE LARGE INTESTINE.—*Pathology.*—In the croupous variety of dysentery, the same changes are found in the large intestine that are met with in croupous inflammation of any mucous membrane. The mucous membrane is congested and swollen, and its free surface is covered with a layer of fibrin, pus, and new connective tissue. Beneath the mucous membrane there is an infiltration of the same inflammatory products. The inflammatory process is not, however, usually evenly distributed over the whole surface of the mucous membrane: it involves patches of it. These become coated in the way I have described, and between the patches the mucous membrane is simply congested. The size and closeness of the patches to each other varies with the severity of the disease, the more severe the disease the larger the patches and the closer they are to each other. After the inflammation has lasted a few days, if there has not been a very large production of fibrin and pus, and especially if these have not been produced in large quantities beneath the mucous membrane, they will degenerate and disappear, and the mucous membrane will return to a natural condition. If, however, there has been a large production of fibrin and pus, and these have been infiltrated beneath the mucous membrane, the affected parts will not return to their natural condition. On the contrary, they will degenerate and be cast off in the same way that the inflammatory products are cast off. It is in this manner that ulcers are formed, and there is a destruction of the mucous membrane. In the less severe cases these ulcers do not extend deeper than the thickness of the mucous membrane. But, if the inflammatory process is more severe, the ulcers will involve the connective tissue coat, or even the muscular coat, or they may extend to the peritoneal coat and perforate through the wall of the intestine. The defects thus produced in the mucous membrane are eventually supplied by cicatricial tissue. The way, therefore, in which patients recover from an attack of croupous dysentery depends upon the intensity of the inflammatory process.

In croupous dysentery, as in catarrhal dysentery, the favorite seat of the inflammation is the lower part of the large intestine, but it is much more common in croupous dysentery for the inflammatory process not to be confined to this part of the large intestine. We meet with a considerable number of cases in which the whole length of the large intestine is inflamed, and occasionally we meet with cases in which only the upper part is involved.

Symptoms.—These are both local and constitutional, and, as in the catarrhal form of dysentery, a certain number of cases are preceded by an ordinary diarrhœa. In others, there is no preceding stage of diarrhœa, but the symptoms belonging to the disease are developed abruptly.

The local symptoms of croupous dysentery are the same as those of catarrhal dysentery, but they are more marked. The number of passages is greater; they may be very frequent indeed, occurring every hour, half hour, or every fifteen minutes, during a considerable part of the twenty-four hours. The passages are composed of blood and mucus, the blood being a more constant factor than in cases of catarrhal dysentery. After the disease has lasted a little time, the patient, in addition to blood and mucus, passes a certain amount of thin, brownish fluid of about the consistency of water. Sometimes floating in this are to be found small fragments of fecal matter; or there may be no passage of fæces, so that patients really suffer from constipation. They do not pass any amount of fecal matter, although they do have frequent passages of blood and mucus and watery fluid. Pain and tenesmus are more marked than in catarrhal dysentery.

The constitutional symptoms are usually a febrile movement and a certain amount of prostration. The febrile movement may or may not be preceded by one or more rigors. The thermometer indicates a temperature of from 100° to 103° in most cases. In bad cases it may indicate a temperature of 105° or 106°. The degree of prostration also varies with the severity of the inflammatory process. The patients are almost always sick enough to stay in bed. Sometimes they are very much prostrated, not being able to sit up in bed. These are most of the constitutional symptoms met with, but in the more severe cases others may be added; the urine may become very scanty or suppressed; the patients may become delirious; they may suffer from convulsions and may pass into a condition of semi-coma or even of complete coma; the tongue becomes dry and brown, the face haggard and anxious; the skin of an unnatural livid color, and in very bad cases the patients pass into a condition of collapse; the pulse becomes exceedingly rapid and feeble, the skin cold, and they finally die in a condition of collapse.

The worst cases of croupous dysentery are seen in tropical countries, where the disease occurs epidemically. They are met with in very young and very old persons.

Symptoms when Lesion is Confined to Upper Part of Colon.—If the inflammatory process is confined to the upper part of the colon, the symptoms will be somewhat different and it will be very easy indeed for the case to pass unrecognized as one of dysentery. These patients do not have the characteristic dysenteric stools. They do not pass blood and mucus, nor do they have tenesmus. They have the same constitutional symptoms as in the ordinary cases of dysentery, but the diarrhœa is of the ordinary variety. There is some febrile movement and some prostration. The febrile movement, as before, may be preceded by rigors. The patients are sick enough to be in bed. They have, however, diarrhœic instead of dysenteric passages. After the disease has lasted for several days, these patients may pass blood from the bowels, and in considerable quantities, as do patients with typhoid fever.

The duration of croupous dysentery is longer than that of the catarrhal variety. It always lasts as long as a week and not infrequently two weeks, and it may last three or four weeks, and sometimes even longer than this. During all this time the febrile movement will not be absent altogether, but there will be exacerbations in the evening. When the disease has lasted two or three weeks, the patients may pass into a typhoid condition; the tongue become dry and brown; there may be low, muttering delirium, and their condition may become pathetic; they will look very much like patients suffering from typhoid fever, and the diagnosis between this form of dysentery which occupies the upper part of the large intestine and the ordinary cases of typhoid fever is sometimes difficult.

Prognosis.—The prognosis is not as good as is that of catarrhal dysentery. A certain number of patients die at the end of a week or later, simply from the effect of the disease. Of the patients who do not die, a considerable number of them recover incompletely. They become the subjects of chronic dysentery. The destruction of mucus membrane may be so extensive that the intestine will never return to a normal condition. This form of dysentery is, then, always to be looked upon with some feeling of apprehension.

Treatment.—As regards treatment, the first and most important thing is to put the patients in bed and keep them there. There is no difficulty in doing this, for they usually feel sick enough to go to bed. As in cases of catarrhal dysentery, if these patients are seen at the commencement of the attack, it is well to begin with a dose of castor oil and opium. Such treatment, however, is not succeeded by a cessation of the symptoms. The patients are better, but the disease continues to run its course. After the first dose of castor oil, if the case is not a very severe one, it may be sufficient merely to give the patients opium, either by the mouth, rectum, or skin, in sufficient quantities to keep them comfortable. Opium may be combined with bismuth or with one of the alkalies, or it may be combined with ipecac. In the milder cases of croupous dysentery, this will be all the treatment that is necessary. In the more severe cases, however, this treatment will not be sufficient. The disease will run such a severe course and the constitutional symptoms will be so marked, that it will be necessary to do something more than this. The drugs which are especially used under such circumstances are ipecac, calomel, ergot, the mineral or vegetable astringents and turpentine. The plan of treatment which has seemed to me most successful in these cases occurring in New York is to begin with a dose of castor oil as before indicated. After this give two or three twenty-grain doses of ipecac according to the severity of the disease and the condition of the patient. In addition, you give at the same time a small hypodermic injection of morphine. After this, nothing more is given except opium in such quantities as will allay the pain and restlessness. After three or four days, if the disease still continues, the same procedure is repeated, beginning with a dose of castor oil and following with twenty-grain doses of ipecac and hypodermic injections of morphine. This may have to be repeated several times during the progress of the case in which a considerable part of the intestine is involved, as it may require several weeks for the disease to run its course. If the heart's action becomes feeble and the disease continues, in addition to these measures, stimulants should be given, either in the form of wines or liquors, in such quantities as will keep the pulse at a fair force. After the disease has lasted four or five weeks, as it sometimes does, castor oil and ipecac are no longer indicated, for by this time the original inflammatory process has run its course, and the symptoms, if any continue, are those due to the ulcers left by the inflammatory process. The patients are no longer suffering from croupous inflammation of the intestine, but from the ulcers caused by this inflammation, and for these ulcers ipecac and castor oil are of no service. At this time the use of the mineral acids, such as dilute sulphuric, nitromuriatic, or hydrochloric acid is indicated; in addition enemata or suppositories may be found beneficial. If suppositories are used, the best drug is iodoform, putting in each suppository as much of the drug as possible, and combining with it a small amount of opium. If enemata are used, the drugs employed may be iodoform, weak solutions of sulphate of zinc or weak solutions of the chloride of zinc.

LECTURE XXXIX.

DISEASES OF THE INTESTINES—CONTINUED.—FOLLICULAR AND CHRONIC DYSENTERY.—CARCINOMA OF THE INTESTINE.

Follicular Dysentery; Pathology, Occurrence, Symptoms, Treatment.—Chronic Dysentery; Pathology, Symptoms, Treatment.—Carcinoma of the Intestine; Varieties, Occurrence, Symptoms.

FOLLICULAR DYSENTERY. *Pathology.—Gentlemen:* We come now to the last variety of dysentery, the so-called *follicular dysentery*, which involves the solitary glands of the large intestine. These glands become enlarged, softened, and break down at their centres; the mucous membrane directly over them is destroyed, and in this way little circular ulcers are formed with overhanging edges; added to this process there is usually a certain amount of acute catarrhal inflammation. In these cases the disease is apt to involve a considerable part of the length of the large intestine.

Occurrence.—It is this form of dysentery which is especially common in armies. We see examples of it in New York, but not very often; it is not nearly as common as the catarrhal and croupous forms.

Symptoms.—The symptoms of this form of dysentery are not exactly like those of the ordinary catarrhal and croupous varieties. The patients not only have the small passages of mucus and blood, which are characteristic of dysentery, but in addition they have loose fecal passages, and in a given case, one or the other may predominate. There is usually a febrile movement, especially at the commencement of the disease, but this lasts only for a short time, or a few days. The diarrhœic or dysenteric passages continue; the patient now begins to emaciate and lose strength. The disease is particularly apt to run a chronic course. After the constitutional symptoms have subsided, the local symptoms may continue for an indefinite period. The patients continue to lose flesh and strength and to have these passages for months, and sometimes for years.

Treatment.—The treatment for this form of dysentery varies with the circumstances under which it occurs. If it occurs in armies, or in soldiers quartered together, the one thing which is most important is, if possible, to send the patients away from camp or the barracks, or whatever place the army may be assembled in. The patients should be sent home, or to a region as remote as possible from the army; this alone is, in some cases, sufficient to bring about a cure. As soon as the patient reaches a place distant from that in which he was attacked he begins to improve, and gradually returns to a healthy condition. In sporadic cases, such as occur in New York, the indications for treatment are much the same as in catarrhal dysentery. If the case becomes chronic, the mineral acids, iron, and quinine should be administered, and, if possible, the patient should travel.

Chronic Dysentery.—We come now to chronic dysentery, which is in most cases the result of acute dysentery, and follows directly upon it; but in some cases the disease is from the beginning a chronic one; there is no acute attack, the patients at once beginning to suffer from the symptoms of chronic dysentery.

Pathology.—The lesions of chronic dysentery vary with the particular form of acute dysentery which it follows. If it follows acute catarrhal dysentery, and the patients finally die from the disease, the lower part, or the entire length of the large intestine is found very much thickened—the whole of the intestinal wall is usually uniformly hypertrophied—and there may be in addition little polypoid tumors projecting into the intestine.

If the disease follows the croupous variety of dysentery a number of ulcers will be found, due to the sloughing of that part of the intestine which has been infiltrated with fibrine and pus. The size and number of these varies. In the worse cases, the whole inner surface of the intestine will be formed of granular tissue, instead of mucous membrane; there is also thickening of the wall of the intestine.

If the disease follows the follicular form of dysentery, ulcers will again be found, but they differ in appearance from those left by croupous dysentery. There are small round ulcers with overhanging edges, and there is a considerable amount of mucous membrane left between them.

Symptoms.—The symptoms vary in these three different kinds of chronic dysentery. If the chronic dysentery follows the catarrhal variety the patient has small passages of blood and mucus, and these may continue for months or years, but not uninterruptedly. There may be days and weeks during which the patients will have no such passages, but after these intervals they will begin to have them again. In addition to passages of blood and mucus, the bowels may be constipated or loose. This seems to depend upon the extent to which the intestine is involved in the inflammatory process. If only the lower end of it is implicated, constipation will exist; but if the whole length of the intestine is involved, the passage will be loose.

If chronic dysentery follows the croupous variety, the patients will first have passages of blood and mucus, and these again will not be continuous; but the amount of blood passed is usually greater than in those cases following the catarrhal dysentery. Again, there may be either constipation or diarrhœa in addition to the dysenteric symptoms proper.

If the disease follows follicular dysentery, the patients usually have diarrhœa without any passages of blood or mucus. It is possible that they may pass, from time to time, a little blood and mucus, but, generally speaking, they simply have a number of loose fecal passages during the twenty-four hours.

In all these three varieties of the disease, while it is running its course, the patients emaciate very much, lose strength, and become unfit for any kind of business. Often they are not sick enough to keep in bed, yet they are miserable. If a sufficient extent of the intestine is involved, the patients do not get well, but finally die from extreme exhaustion, or from some intercurrent disease. If only a small part of the intestine is involved, it is always possible for these patients to get well after the lapse of a considerable length of time.

Treatment.—The treatment of these cases is partly general and partly local. The local treatment consists in applications to the rectum of drugs which will exert an influence upon the inflammatory process. Drugs may be applied to the rectum in the form of enemata or suppositories. If enemata are used, simple cold water, or dilute solutions of the chloride of zinc or the sulphate of zinc, or iodoform may be used. If iodoform is selected, it may be used alone, or in combination with a little bismuth or a little opium.

In cases of chronic croupous dysentery the same enemata or suppositories may be used, but in addition the patient may be placed under the influence of ether, a Sims' speculum introduced into the rectum, and if the ulcers are sufficiently near the anus remedies may be applied directly to them. For this purpose strong solutions of nitrate of silver, or the solid stick of nitrate of silver, or strong nitric acid, or carbolic acid may be used. If the ulcers are not sufficiently low in the rectum, or the patient will not submit to this treatment, you are to go on treating the case as before. In addition to these local means, constitutional treatment is to be used. This is of two kinds: first, it may be directed toward the diet; and second, toward the general health of the patient. As regards the food, these patients may be put upon a simple milk diet and confined entirely to it. This plan is of a great deal of service in the treatment of a certain number of cases, especially those of the catarrhal variety, in which there are no ulcers. In this way some of these patients may be entirely cured. To improve the general health of the patient, the ordinary means, such as tonics, mineral acids, iron, quinine, and strychnine may be used. If possible, the patient should live in a warm climate, where he can get out of doors and breathe fresh air.

TUMORS OF THE INTESTINE.

CARCINOMA OF THE INTESTINE.—*Varieties.*—We go on to the consideration of the new growth (*tumors*) which we find in the intestine. The most common of these is cancer—primary *carcinoma.* Carcinoma of the intestine is found in one of three ordinary anatomical shapes. Either in the form of simple cancer, where there is a collection of polygonal cells enclosed in cavities surrounded by connective tissue-stroma, or it is found in the form of colloid cancer, or in the form of a new-growth composed of tubes or follicles lined with cylindrical epithelium, and imbedded in the stroma of the connective tissue.

Site.—The new growths are situated almost always in the large intestine, but it is possible for them to be situated in the small intestine. In the large intestine they are met with in one of three positions: either in the caput coli, lower part of the rectum, or anus. When they project from the anus, they seem to be merely new growths of the skin about the anus, and not growths of the rectum proper. And yet, from their situation, occupying the opening of the intestine, they produce constitutional symptoms, and are usually considered as new growths of the large intestine. They project both upward into the cavity of the rectum, and outward by the anus in the form of fungous masses. When the disease occurs in the caput coli, or when it occurs in the lower part of the rectum, it begins in the wall of the intestine, and usually surrounds a certain part of it. It does not usually extend very far along the length of the intestine. It may only occupy an inch, or two inches; sometimes three or four inches along the length of the intestine.

After the new growth has infiltrated the wall of the intestine an ulcerative process is usually set up, which is accompanied by the destruction of a certain part of the mucous membrane which covers the new growth. In addition it is found that the new growth projects inward into the cavity of the intestine. The projecting mass is sometimes in the form of fungus or irregular lobulated tumors, which are sometimes hard, sometimes soft. At times the projection is more symmetrical, so that there is a sort of septum formed in this part of the gut, there being a narrowing of the calibre of the canal at this point. In other cases the new growth does not project into the intestine. The intestine may be dilated instead of narrowed. There is always a certain amount of local peritonitis, which begins in the peritoneum covering the intestine at the point where the new growth develops. As a result of this adhesions are formed, and little collections of pus may be shut in by them. In some cases the inflammatory process (peritonitis) will become more general, so that the intestine is matted together throughout the whole of the abdominal cavity. In a certain number of cases the ulcerative process progresses until perforation of the intestine takes place. If there are no adhesions at the point of perforation, the contents of the intestine escaping into the peritoneal cavity sets up a peritonitis which usually proves fatal. If there are adhesions, the contents of the intestine escape little by little, and usually there is a small abscess shut in by the adhesions. If situated posteriorly the abscess may burrow and form a cavity of considerable size, which will be filled partly with pus, partly with a dirty fluid, and partly with fecal matter.

Occurrence.—In New York and the country about, the disease is tolerable common. It occurs during adult life and old age; examples of it are very seldom seen in persons younger than thirty years old.

Symptoms.—The symptoms of cancer of the colon or large intestine will vary with the position of the new growth. If the new growth is situated in the rectum it is usually a few inches above the anus, or just within reach of the finger when introduced into the anus. After the new growth has existed for a while it seems to descend by its own weight, so that when an examination is made, it is usually found within three inches of the anal orifice. The symptoms of cancer of the rectum usually begin very slowly and insidiously. At first the patients are not at all aware that they are suffering from a mâlignant disease. They think they are suffering from some disturbance of the digestion or the bowels. The bowels are constipated and flatulent. The constipation continues to become more marked, and the patients begin to notice that the fæces have an unnatural appearance. They are not of the normal size and shape. They pass solid fæces in the form of small cylinders or ribbons. At this time there is commonly added a certain amount of pain, which is referred to the region of the rectum. The patients begin to emaciate and lose strength. They become pale and cachectic. The constipation continues. The characteristics of the fæces become more marked. The patients depreciate more and more, until finally they succumb to the disease. When these patients apply for treatment it is, as a rule, not until the new growth has lasted for a considerable length of time and has attained some size; hence, there is commonly no difficulty in making the diagnosis. As soon as the finger has passed up the rectum it comes in contact with these tumors, which are sometimes hard and nodular. Sometimes, however, they are so high in the rectum as not to be readily reached with the finger. Under such circumstances it is necessary to etherize the patient and dilate the rectum, when the finger can be introduced further, and if it is necessary, the whole hand may be introduced into the rectum. The tumor usually presents in the form of a soft fungous mass, which projects into the gut. It is so soft that it can be broken down with the finger; at other times these tumors feel like hard nodular masses. In other cases the feeling imparted to the finger is that of a septum of hard new tissue completely stopping the gut; at other times the calibre of the rectum is about the natural size, but a hard, firm, immovable new growth is felt in the wall of the rectum and around it.

In other cases the disease does not begin in exactly this way. The first symptom, instead of being constipation, is an irregular diarrhœa.

From time to time the patients have a number of loose fecal passages from the bowels. At other times they have small passages of blood and mucus. They experience a feeling of tenesmus or irritation, which makes them feel as if they were about to have a passage from the bowels, and when they try to do so they pass nothing except a little mucus and blood. These patients not infrequently think they are suffering from piles, owing to the uncomfortable feeling which they experience about the lower rectum. After a longer or shorter time, pain comes on, and it varies as to its severity. These patients lose flesh and strength ; they become anæmic and cachectic, just as did the first set of patients. When they apply for treatment, a tumor is felt as before, but it is not, as a rule, of such a character as to obstruct or close the calibre of the gut. The new growth takes place around the intestine and does not prevent the escape of fæces from the bowels. In both sets of cases there may be paralysis of the sphincter muscle of the anus, and in nearly all cases the patients are no longer able to contain their fæces. They pass them involuntarily, and, in addition, there is a constant dribbling from the anus of a brownish, bloody fluid, which is apt to irritate the skin and may produce ulceration of the skin in this situation. In some cases the new growth extends to the bladder, or if it is in a female, it may extend to the vagina. When this happens, there are bladder symptoms, or a communication may be formed between the rectum and vagina (recto-vagina fistule). The local symptoms become more marked and the general health more depreciated until finally the patients die. A certain number of them die simply worn out by the disease ; others seem to die from the accumulation of fæces in the large intestine, which may under such circumstances become enormously distended. In other cases the patients die from rupture of the intestine, which takes place either by ulceration or from distention of the intestine by the fæces. Again, death may be hastened by a general chronic peritonitis which is set up, for, if the disease extends to the bladder and involves the openings of the ureter, there may be retention of urine produced in this way and the patients may die from the condition of the kidney ; from hydronephrosis. Patients usually die within a year from the time that the existence of the disease is fairly made out. You may usually feel pretty sure of your statement in telling the friends that the patient will die within a year from the time you are positive of your diagnosis.

LECTURE XL.

DISEASES OF THE INTESTINES—CONCLUDED. —TUMORS OF THE INTESTINE.

Carcinoma of the Rectum; Treatment.—Carcinoma of the Caput Coli; Pathology, Symptoms, Treatment.—Lymphoma of the Intestine; Pathology, Occurrence, symptoms, Prognosis, Treatment.

CARCINOMA OF THE RECTUM.—*Treatment.—Gentlemen:* The treatment of carcinoma of the rectum, of course, is palliative. In the larger number of cases, when the tumor is situated very low in the rectum and is still circumscribed, it may be removed entirely by surgical procedure. This may prolong the life of the patient for a considerable length of time. When, however, the disease is higher in the rectum, and there is a diffuse infiltration into the surrounding soft parts, surgical interference is of no avail; the only thing to be done is to administer opium, in the form of suppositories or by the mouth, in sufficient quantities to ease the pain.

Carcinoma of the Caput Coli.—When the new growth is situated in the caput coli, the symptoms are again, at first, insidious and slow in making their appearance. In some of the patients the first symptom that is noticed is that their general health is beginning to fail. They have no particular disturbance of the bowels. There may be a certain amount of flatulence, but there is no diarrhœa or constipation. The patients find that they are losing strength and flesh and becoming anæmic. There may be nausea and vomiting, so that, at first sight, the organ at fault seems to be the stomach instead of the large intestine. After a time a tumor is formed in the situation of the caput coli. Sometimes the patients discover this themselves; at other times it is not discovered until they seek medical advice, and in still other cases it is not discovered until after death. When these tumors are felt through the abdominal wall, they feel like pretty hard circumscribed bodies. They are not movable to any extent. They may be moved from one side to the other, but it is always evident that they are attached to the posterior abdominal wall. In many cases they are not very large, feeling through the abdominal wall as if they were about as large as a pigeon's egg or that of a hen, or sometimes a little larger than this. Occasionally the intestine above the caput coli (the small intestine) is distended with fæces, and under such circumstances a larger tumor is produced, the outlines of which are not as distinct. The feeling imparted is that of impacted fæces, which it really is. The tumor is partly made up of new growth and partly of an accumulation of fæces in the small intestine. At times it is very difficult indeed to determine whether the patient is simply suffering from impaction of fæces, or whether the impaction of fæces is due to a new growth in the wall of the intestine.

In other patients, the first symptom noticed is pain referred to the region of the new growth. This pain, however, may, at first, not be very characteristic; it may resemble that of ordinary intestinal colic. The patients will tell you that, from time to time, they suffer from a severe attack of pain in the lower part of the belly, upon the right side, which lasts for a few minutes, hours, or days, and then disappears and may not return again for some little time. With the pain there is most commonly a certain amount of flatulence, and these may be all the symptoms complained of. The colicky pain may be considered by the patients as due to articles of food that they eat, for they find that the pain is brought on more frequently by eating some particular food, and this again tends to mislead them and the physician into the belief that it is some disorder of the stomach or intestine. After a time, however, the tumor makes its appearance. The tumor is the same in either set of cases, and may be increased in size by an accumulation of fæces from above. In both sets of cases, after a time, the patients lose flesh and strength, and the characteristic cachexia becomes so marked that there can no longer be any question as to the fact that they are suffering from malignant disease, and, under such circumstances, if you can feel a tumor the diagnosis is set at rest, but until you feel a tumor the diagnosis is always uncertain. In addition to these symptoms, the patient may suffer from constipation or diarrhœa, which depends upon the way in which the new growth is developed. . If it is developed so as to partially fill the cavity of the intestine, there will be an accumulation of fæces above it, or in other words, constipation. If, on the other hand, the new-growth is developed in such a way that the cavity of the intestine is not occupied by it, there will be no constipation, and instead there may be an irregular diarrhœa.

Treatment.—These patients become worse and worse, just as do those suffering from cancer of the rectum. There is no surgical interference which can afford any relief. The only thing that can be done is to palliate the pain from which these patients suffer. The most efficient of all drugs is opium, and it is always to be used in the latter stages of the disease. The pain varies as to the time in the course of the disease at which it begins, and also as to its severity. In the earlier stages of the disease, when the pain is not constant, good results will sometimes be obtained from the use of the different cordials. These are of more service than either brandy or whiskey. Sherry cordial, or any of the different cordials will answer for the relief of the pain. Of course, the use of opium should be postponed as long as possible. As soon as the pain cannot be controlled in any other way, opium is then to be used.

Lymphoma of the Intestine.—Besides carcinoma of the intestine there is another form of new growth, occurring not very frequently.

Pathology.—It has the structure of an ordinary lymphatic gland, and for this reason it is called by the name of lymphoma. This form of new growth involves either the small or large intestine; it is not as regularly confined to the large intestine as is carcinoma. The new growth, although finally involving the wall of the intestine, does not begin there, but in one of the lymphatic glands of the mesentery in the immediate neighborhood of the intestine. Beginning here, it increases in size, and finally involves the wall of the intestine, which becomes infiltrated with a new growth which is composed of lymphatic glandular tissue. The new growth, in the greater number of cases, surrounds the wall of the intestine, and, in some cases, projects into its cavity, so as to obstruct the passage of the fæces through it. In other cases there is ulceration, which destroys a part of the wall, so that the intestine at this point becomes larger than normal. In this, as in carcinoma, there is often a local peritonitis, which results in the formation of adhesions, and there may be little collections of pus shut in by these adhesions. Again, as in carcinoma, there may be an ulcerative process established, which will result in the perforation of the wall of the intestine and give rise to abscesses in the abdominal wall which connect with the wall of the gut, and in consequence of this will contain a certain amount of fecal matter. Such cases may be mistaken for perityphlitis and the patients be operated upon for the relief of this, and even during the course of the operation a mistake is not made out. The surgeon cuts down upon an abscess, but the patient, instead of getting well, as is usually the case after the opening of a perityphlitic abscess, dies, and it is then ascertained that the abscess is secondary to a new growth in the wall of the intestine.

Occurrence.—There seems to be no particular age at which the disease is especially liable to occur. It occurs in young persons as well as in old; but whether it ever occurs in children, I do not know.

Symptoms.—The symptoms of this form of new growth are very much the same as those of carcinoma of the caput coli. The patients lose flesh and strength. There may be disturbances of the stomach: nausea, and vomiting. Pain may exist and be referred to the situation of the new growth. There may be constipation or diarrhœa. When the new growth has reached a sufficient size, it can be appreciated by palpation; but, after finding this, you are not as certain of your diagnosis as in cancer of the colon. In the latter affection a tumor is found either in the caput coli, or rectum, but lymphoma may be situated in any part of the abdominal cavity. More than this, when the tumor occurs in the wall of the small intestine, the weight of it, as a rule, is sufficient to drag it down, so that the tumor will not be situated in the normal position of the intestine from which it grows. I have seen such a new growth of the duodenum found in the region of the caput coli, and it was mistaken for a growth of the caput coli, and even at the autopsy it took some little tact to make out its real location.

Prognosis.—The prognosis is as bad as in cancer of the intestine. Some patients die worn out from the disease, some from constipation, others from peritonitis, and still others from prostration.

Treatment.—The treatment is the same as that for cancer of the caput coli, the object being simply to relieve the pain from which the patients suffer, knowing that life will not be prolonged for a great length of time.

LECTURE XLI.

DISEASES OF THE LIVER.—FUNCTIONAL DISORDERS.

Size and Position of the Liver; Functions.—Functional Disorders of the Liver; Two Principal Varieties; Etiology, Symptoms, and Treatment of Each.

SIZE AND POSITION OF THE LIVER.—*Gentlemen:* We go on now to the consideration of diseases of the liver, and, in the first place, I must call your attention to the normal position of that organ, and to the way in which we determine changes in its size and position. The liver is, as you know, situated in the right hypochondriac region, extending across the abdomen to the left, so as to occupy the epigastrium and to project for a little distance into the left hypochondrium. When we wish to determine the size of the liver, we try to make out its upper and lower borders. In a normal adult who has a healthy liver, the upper border of it in front ought to be situated at the lower edge of the fourth rib or the lower edge of the fifth rib. This amount of variation is found in healthy individuals. When we examine for the upper edge of the liver in the axillary region, we will find it situated lower than in front; it corresponds to the seventh rib. The upper border of the liver posteriorly we are usually unable to make out, owing to the fact that it is covered by so much lung. This is best seen in a vertical section of the liver and lung, which will show plainly the extent to which the lung overlaps the liver posteriorly. The lung also overlaps the liver in front down to the sixth rib. The fact that the upper part of the liver is covered by lung and the lower part is not so covered makes a difference in the percussion note which is obtained. We only expect to get dulness over the upper part of the liver down to about the sixth rib, and the degree of dulness depends upon the condition of the lung; if the patient is emphysematous, the dulness will be less marked than if the lung is in a normal condition. In all cases the sound obtained by percussion is dulness; it is not flatness. When, however, we percuss below the sixth rib we will find flatness as far down as the free border of the ribs. The flatness thus obtained is also modified close to the free border of the ribs by the condition of the intestine in the abdominal cavity. If the intestine is distended with gas, the flatness will not extend fairly down to the free border of the ribs, even if the liver is normal, but if the intestine is empty, we will get flatness down to this point. In the axillary region the liver is not overlapped by lung, and hence we get flatness at its upper border. Behind we are unable to obtain liver dulness upon percussion, on account of the extent to which it is overlapped by the lung tissue. In front the lower border of the liver ought to extend to the free edge of the rib pretty exactly. We try to make it out in two ways, partly by percussion and partly by palpation. In determining this point by percussion, we percuss downward until we find the point at which we fail to get flatness and begin to get tympanitic resonance. This point ought to mark the lower border of the liver, but in practice you will find great difficulty in determining it by percussion, owing to tympanitic resonance. Hence it is necessary to resort to palpation. The lower border of the liver may be determined by palpation in two ways. The patient ought always to be laid flat upon his back in bed, the fingers should be placed upon the abdomen, and with them you should make a quick gentle pressure over the region of the liver. By this means, if the liver is below the free border of the ribs, it will, in a certain number of cases, be very distinctly felt; in other cases the abdomen will be so much distended by gas in the intestine, or the wall of the abdomen will be so thickened with fat, that you cannot make it out by this quick, gentle palpation. Under such circumstances, it is necessary to use firm, steady pressure, and in doing this it is usually necessary to use both hands. While pressing, you cause the patient to take a deep breath and let it out again. When the patient is doing this, you crowd the fingers inward and up under the lower edge of the ribs, and in this way you can usually make out the lower border of the liver. There is hardly any other point in physical diagnosis so difficult to determine as the lower border of the liver. A mistake is often made in interpreting the sensation given by the abdominal muscles as due to something in the abdominal cavity. The lower border of the liver should, as I have said, be on a line with the free edge of the ribs, except in the epigastrium, where it comes down an inch or an inch and a half or two inches below the cartilages of the ribs.

Functions of the Liver.—We have also to remember some points in regard to the natural functions of the liver. The liver seems to perform three principal functions. In the first place, there is formed in it a substance called glycogen, which appears to contribute to the nutrition of the body, and to be concerned in the formation of the white globules of the blood. In the second place, the liver effects changes in the blood which circulates through it. The substances thus affected are of an albuminoid character, and are changed into two sets; one set aids in the nutrition of the tissues, and another set is excreted by the kidneys in the form of excrementitious matter. In the third place, the liver liberates a fluid of a peculiar composition, called bile. The bile, when formed, passes from the liver by means of the bile-ducts into the small intestine. After reaching this point a certain part of it seems to assist in the digestion of the fatty matters and the peptons contained in the small intestine. Another part of the bile seems to stimulate the peristaltic action of the intestine and to prevent the decomposition of the fæces, but the largest part of the bile is reabsorbed from the small intestine.

You will observe, then, that the liver, so far as its functions are concerned, is one of the most important viscera of the body, and the functions which are especially important are the assistance which it renders to digestion, and the separation of food which has been absorbed from the intestine and stomach into substances fit for the nutrition of the body and substances which are excrementitious and require removing from the body.

FUNCTIONAL DISORDER OF THE LIVER.—The functions of the liver may be disturbed, and for this reason there are certain *functional disorders of the liver.* These may be associated with structural changes in the liver, or there may be no such changes, as far as we are able to determine by any means with which we are acquainted.

Etiology.—Functional disturbances of the liver are very common. They belong principally to two different classes. In the *first* set of cases the disturbance of the functions of the liver is related to the production of bile; it is the bile-producing function of the liver which is interfered with. In the *second* set of cases there is no disturbance of the production of bile. It is produced in sufficient quantity and of proper quality. The function which is disturbed is the normal change of the nutritive matters which are held in solution in the blood, and which pass through the liver. These, instead of undergoing such a chemical change that a part of them can be used for the nutrition of the body and a part excreted, do not undergo these changes, and as a result of this, marked symptoms are produced. Instead of holding in suspension substances which can aid in the nutrition of the body, substances are held in suspension which are not used for this purpose, and which cannot be contained in the blood without producing abnormal symptoms.

Symptoms in the First Set of Cases.—As a result of the interference with the bile function, patients become badly nourished, thin, pale, and emaciated. They suffer very frequently indeed from headache and from curious nervous symptoms of different kinds—pains of an irregular character in different parts of the body. They are often sleepless. They may become hypochondriacal and anxious about themselves. There may be a functional disturbance of the action of the heart, liver, or almost any part of the body, and these are often of the most irregular and fanciful character. The bowels are always constipated, hardly ever acting without the use of medicine. Flatulency is marked, the patient constantly belching up wind from the stomach. The urine is unchanged, or it contains an undue amount of the oxalate of lime or of the phosphate of lime. We meet with all degrees of suffering from such a functional disturbance of the liver. In some, the symptoms are comparatively trivial, the loss of general health not being marked, the nutrition tolerably fair, the most prominent symptoms being the nervous ones, flatulency and constipation of the bowels. In other patients, however, where the disturbance is more marked, the general health will suffer a good deal. These patients will become so emaciated that it is often very difficult to tell whether or not they are suffering from some important organic disease.

Treatment.—In treating these patients there are two special indications to be borne in mind. In the first place, to increase, if possible, the production of bile and try to restore the function of the liver, which is interfered with. In the second place, to improve the general nutrition and health of the patient as much as possible, and in this way bring about an improvement in the condition of the liver.

Unfortunately, our knowledge is not very exact as to what drugs really increase the amount of bile which is produced by the liver. The most exact information that we have on this point is from experiments carried on by Prof. Rutherford, of Edinburgh. These experiments were made upon dogs, and so far as dogs are concerned were quite satisfactory; but we can never be certain that the effect of drugs upon human beings is the same as upon animals, hence the best that we can do at present is to use those drugs which are found to be most efficacious in increasing the production of bile in dogs. Of such substances, podophyllum in small doses is the best. It was found, although small doses of this drug increased the production of bile, yer, when it was given in large doses, it diminished its production. Aloes, also, was found to increase the production of bile, but for this purpose it must be given in large, and not in small doses. The same is true in regard to rhubarb. If rhubarb is given to increase the production of bile, it must be given in pretty large doses. Ipecac increases the production of bile, and it can be given in small doses; large doses are not necessary. Dilute muriatic acid is another drug of considerable efficacy. The phosphate, benzoate, and salycilate of sodium increase the production of bile. The phosphate of ammonia and the bichloride of mercury in small doses also have a decided effect in the same direction. This furnishes us with a list of drugs which we may use in such cases as we are now considering, with a rational expectation that one or other of them will increase the amount of bile, and in this way the condition of the patient will be improved.

In selecting a drug for this purpose you will have to be guided by its effect upon the particular patient under treatment. Sometimes one will be found to answer best, sometimes another, and you will use whichever one is best borne by the patient.

In order to improve the general condition of the patients, they should be fed as well as possible. They should be required to take as much solid and nutritious food as they are able to digest, and they should also have either wine or liquor in such quantities as are necessary to improve digestion. If it is possible for the patients to travel, you will have them do so. The constipation can usually be relieved by means of one of the drugs which are of use in increasing the production of bile. For the nervous symptoms, if they are very prominent, it is often necessary to give one or other of the bromides, or chloral hydrate, guarana, or assafœtida; these nervines should be given as little as possible, and opium is never used, no matter how much pain the patient may be suffering.

Second Set of Cases.—We come now to the second set of cases of functional disturbance of the liver, the cases in which the production of bile is not interfered with, but in which the condition of the substances that pass through the liver is not the same as normal. Patients thus affected do not become emaciated, but, on the contrary, they are usually stout, florid, robust-looking persons. They are larger, fatter, and more muscular than average individuals. They are persons who eat and live well, and who indulge in the use of alcoholic drinks and tobacco. They are people who live in this comfortable way, not taking exercise in proportion to the stimulants which they use.

Symptoms.—The most prominent symptoms are the nervous ones, especially those referred to the head. Patients very often complain of vertigo or dizziness, and the dizziness may become very marked indeed; so much so that patients may fall to the floor, or they may become unconscious for several minutes. Sometimes, when the vertigo is not as marked as this, it will be sufficient to distress the patients considerably. They will be afraid to go up or down stairs without taking hold of the baluster, and when they look down for some distance they are always conscious that they are liable to become dizzy and fall. Not infrequently they complain of loss of memory, that they can no longer attend to business as well as formerly, that they have an uncomfortable feeling in the head, a feeling of oppression, as if something was interfering with the cerebral function. They become very anxious about themselves, and think that they will have apoplexy, or that they are becoming insane, and it is often very difficult to convince them to the contrary. As a rule, the bowels are regular. They are not constipated, but, on the contrary, there may be looseness of the bowels from time to time. The urine always contains an increased amount of urates, either red or white, or an increased amount of uric acid, these forming a deposit which is sometimes very abundant. The patients look into the chamber and find an unnatural amount of sediment, and this, too, alarms them. These patients always feel better for a time after having taken a large dose of purgative medicine, and on finding this out, many of them get into the habit of taking, from time to time, some medicine which will cause the bowels to act freely. For this purpose they are very apt to use some one of the quack purgative pills, and to keep them on hand. In this way patients will go on for months or years, during which time they will look well-nourished, and yet they will continue to be miserable and to worry a great deal about themselves, more than any other class of patients you will meet with.

Treatment.—The treatment, if it is to be effectual, will be principally hygienic. There is no particular advantage to be gained by purging the patients, for as soon as the effect of the purge has passed off the symptoms will return. The diet should be regulated. They should not be allowed to take any wines or liquors whatever. They should be compelled to give up the use of tobacco altogether, and not be allowed to eat a very large amount of animal food. Their food should be of a kind that is easily digested, and they should not eat a great deal at a time. In New York, many business men, after eating breakfast, eat nothing else during the day until their dinner hour, which is six or seven o'clock in the evening, when they eat a very large amount of food. This habit should be broken off entirely, and the patients should be obliged to take three meals a day, each of which is sufficient but not excessive. It is also important that these patients should take a great deal of exercise. It is not sufficient for them to take a moderate walk every day, but they require much more violent exercise. They should go into a gymnasium, or walk fast for a long distance, or ride upon horseback. If you can get them to carry out such a mode of life as this, most of them will do very well indeed. They improve gradually and permanently. But it is always difficult to get them to carry out such a plan of treatment. In the first place, they are not likely to believe what you tell them. They would rather take medicine for the cerebral trouble than to change their habits of living, as I have indicated. If any drugs are necessary, muriatic acid, or one or other of the alkalies will be all that is required.

LECTURE XLII.

DISEASES OF THE LIVER—CONTINUED.—JAUNDICE.—ABSCESS OF THE LIVER.

Jaundice; Varieties.—Etiology of Obstructive Jaundice.—Non-obstructive Jaundice; Occurrence.—Theory of the Production of Non-obstructive Jaundice.—Simple Jaundice; Etiology, General Symptoms; Symptoms of Jaundice Due to Catarrhal Inflammation of the Stomach, Duodenum, and Bile-duct; Symptoms of Jaundice Due to Catarrhal Inflammation of the Duodenum; Symptoms of Jaundice Due to Catarrhal Inflammation of the Bile-duct; Symptoms of Jaundice in Patients Previously the Subjects of Fatty Infiltration of the Liver; Treatment.—Abscess of the Liver; Anatomical Varieties.—Idiopathic Abscess of the Liver; Pathology, Occurrence, Etiology, Symptoms, Duration, Physical Signs, Diagnosis, Treatment.

JAUNDICE.—*Gentlemen:* We come now to the consideration of another condition dependent upon the state of the liver, and that is *jaundice.* By this we mean that condition in which the skin becomes stained with the coloring matter of the bile, so that it appears of a yellow or brownish-yellow color, or sometimes of a greenish-yellow color.

Varieties.—We find that jaundice is of two kinds, obstructive and non-obstructive. By obstructive jaundice is meant that which is dependent upon some obstruction to the passage of the bile from the liver to the small intestine. In non-obstructive jaundice there is no such obstruction to the passage of the bile. Obstructive jaundice is more common.

Etiology of Obstructive Jaundice.—The mechanical obstructions to the escape of the bile from the liver into the duodenum are of various kinds. In the first place, there may be foreign bodies in the biliary passages, in the hepatic duct or in the common bile-duct. These are sometimes biliary calculi, sometimes thickened bile, not sufficiently thick to form calculi, yet thick enough to make a solid mass. Sometimes an excess of mucus, less frequently parasites find their way into the bile-duct from the liver or from the small intestine. Foreign bodies in the small intestine may find their way back into the common bile-duct and completely or partially occlude it. Another cause of such interference is inflammation of the bile-duct or of the duodenum. If the hepatic duct, the common bile-duct, or the duodenum in the immediate neighborhood of the opening of the bile-duct become inflamed, there will be swelling of the mucus membrane, resulting in the partial or complete obstruction to the passage of bile from the liver into the intestine. Strictures and stenoses of the bile-ducts are also met with as a result of perihepatitis, or in consequence of ulcers of the duodenum or bile-ducts. Occasionally new growths are formed in the walls of the bile-ducts or in the walls of the duodenum close to the opening of the bile-duct, and these will press upon the bile-duct so as to obstruct the passage of the bile. Again, any large tumors in the abdominal cavity may press upon the bile-duct and in this way produce obstruction. Tumors of the liver, lymphatic glands, stomach, pancreas, or kidneys, or the impaction of fæces may be sufficient to produce pressure upon the bile-duct and give rise to jaundice.

The way in which jaundice is produced in these cases is that the bile accumulates in the biliary passages, and not being able to pass through the common bile-duct, is reabsorbed and circulates in the blood, thus staining the tissues.

Non-obstructive Jaundice.—In the second form of jaundice there is no such mechanical obstruction to the passage of the bile from the liver into the intestine. There is usually no change in the bile-ducts whatever. They are not inflamed and are perfectly pervious; the bile readily passes through them into the small intestine.

Occurrence.—We very often meet with this sort of jaundice as a complication of infectious diseases. A very marked example is to be found in yellow fever. It is also seen in remittent fever, less frequently in typhoïd, typhus, and relapsing fevers; occasionally in measles and sometimes in acute pachymentous inflammation of the liver (yellow atrophy). Some of the mineral poisons, especially phosphorus, mercury, copper, and antimony, will produce jaundice. The inhalation of chloroform and ether will produce jaundice in a few persons. Some cases of concussion of the brain are associated with jaundice. Thrombosis of the portal vein will quite frequently give rise to it.

Theory of the Production of Non-obstructive Jaundice.—There has always been considerable difficulty in understanding how this kind of jaundice is produced; how it is that when the bile passes freely from the liver into the intestine that its coloring matter gets into the blood and patients become jaundiced. A number of explanations have been offered to account for it; perhaps the explanation of Murchison is the most plausible. He tells us that in healthy persons a large part of the bile is regularly reabsorbed from the intestine into the blood, and that after being reabsorbed it undergoes certain changes, so that it does not circulate in the blood as bile. He teaches us that when this form of jaundice occurs, the bile, after being reabsorbed from the intestine and taken into the blood, does not undergo the same changes as in healthy individuals, but remains as bile in the blood, especially the pigment part remains unchanged, so that the bile pigment circulates in the blood and in this way reaches the different tissues of the body and stains them.

In both the obstructive and non-obstructive forms of jaundice, the presence of the coloring matter of the bile in the blood does not seem to be dangerous to the health of the patient. It seems to be compatible with the proper performance of all the functions of the body. If the amount of bile circulating in the blood is considerable, we do have in some cases symptoms accompanying the jaundice, but these are due to other causes. They are not due to the presence of the biliary coloring matter but to some other conditions which are associated with the production of jaundice.

SIMPLE JAUNDICE.—*Etiology.*—Of the forms of obstructive jaundice, there is one variety which is called simple jaundice. The name is not a good one, but it is pretty well established that by this we mean those cases in which there is an acute catarrhal inflammation of the common bile-duct alone, or of the duodenum and common bile-duct, or of the stomach, duodenum, and common bile-duct. In these cases we have the obstructive variety of jaundice, and as a result of the inflammatory process, the mucous membrane of the common bile-duct is thickened to such an extent as to prevent the bile from escaping into the small intestine as it should. These, properly speaking, are cases of gastro-duodenitis, or duodenitis, or inflammation of the common bile-duct. There is in reality an inflammation of the common bile-duct with or without an inflammation of the duodenum and stomach. As a result of this jaundice occurs, and it is customary to call these cases of simple jaundice, instead of by the name which really belongs to them.

General Symptoms.—In cases of jaundice of this kind, patients suffer from a feeling of general *malaise*—they feel dull, heavy and stupid. They are disposed to sleep during the day time, and they very often have a certain amount of headache, or instead of headache, they have vertigo. There may be a disturbance of the stomach, nausea and vomiting. The bowels are usually constipated and the fæces which the patients pass are of an unnatural color. They are not stained by the bile as in health, but are of a white, brownish, or grayish color, or clay-colored, as they are called. The urine, on the other hand, is more deeply colored than normal. It contains bile pigment and is of a deep brown color. There may be a febrile movement, but it is, as a rule, not very marked. Usually, there is considerable pain referred to the region of the epigastrium. Very soon after this jaundice makes its appearance. There are in the different cases some variations as to the exact time when jaundice occurs, and also when the constitutional symptoms are manifested. In some cases, the constitutional symptoms come first. There will be nausea, vomiting, headache, and certain general dulness of the patient for twenty-four, thirty-six, or forty-eight hours before the jaundice makes its appearance. In other cases, the jaundice will be the first symptom. The patient will feel perfectly well, and some of his friends will tell him that he is becoming yellow, and after the jaundice develops itself the constitutional symptoms make their appearance. When the jaundice is developed there is very often a certain amount of itchiness of the skin, and the patients want to scratch themselves all the time. The pulse may be somewhat slower and fuller than it should be. These are the general symptoms of simple jaundice, but they may vary with its exact cause, whether the jaundice be due to the involvement of the bile-duct alone or the bile-duct and duodenum, or the bile-duct, duodenum, and stomach. In addition, we have to distinguish another set of cases, namely, those patients who are the subject of fatty infiltration of the liver previous to the attack of jaundice.

Symptoms of Jaundice Due to Catarrhal Inflammation of Stomach, Duodenum, and Bile-duct.—In the first place, of the cases in which the stomach, duodenum, and bile-duct are all the seat of catarrhal inflammation. In these there are pretty marked constitutional symptoms in addition to the jaundice. The patients vomit a great deal, a febrile movement is usually present to such an extent as to be fairly noticeable, and it may last for several days, as long as a week, or even longer. The patients will be very dull and considerably depressed, and there may be even a certain amount of prostration. They may be sick enough to be in bed part of the time.

Symptoms of Jaundice Due to Catarrhal Inflammation of Duodenum.—In those cases in which the duodenum is alone involved, the constitutional symptoms are not as marked. There may be no vomiting or nausea, or there may be nausea without vomiting; if there is vomiting, it is not a prominent feature. There may be no febrile movement, but if it exists, it is neither marked nor of long continuance. In these cases, pain is apt to be a prominent feature. Pain is also complained of in those cases in which the stomach is involved, but patients are more apt to speak about it when only the duodenum is affected.

Symptoms of Jaundice Due to Catarrhal Inflammation of the Bile-duct.—Cases in which only the bile-duct is involved are the mildest of all. There is usually no disturbance of the stomach, no febrile movement, and the patients are not sick enough to be confined to bed. They may feel a little dull and drowsy, but they are apt to be well enough to attend to their business. There may be no pain whatever, or there may be a little pain. The jaundice may be almost the only symptom which attracts the attention of the patient.

Symptoms of Jaundice in Patients Previously the Subjects of Fatty Infiltration of the Liver.—In those cases where there is fatty infiltration of the liver before the jaundice is developed, the symptoms are particularly marked, and the inflammatory process involves the stomach, duodenum, and bile-duct. Vomiting is a prominent symptom, as well as the febrile movement and prostration. Pain is sometimes severe, at other times not. In addition to these symptoms, we have others which are not found in ordinary cases of jaundice. There are cerebral symptoms. The patients become delirious, and the delirium, after a time passes into semi-coma or almost complete coma, and the patients become more and more prostrated. The febrile movement and cerebral symptoms continue, and the patients pass into a typhoid condition, with a dry tongue, with sordes of the mouth and lips, and finally they die from the disease. These are the only cases in which death ensues as the result of simple jaundice. In other cases, the patients regularly get well after a longer or shorter time.

Treatment.—In the treatment of these cases you must think of what you have to treat. You are not called upon to treat the jaundice, but to treat the lesion which causes the jaundice; you do not wish to cause the liver to secrete any more bile. The trouble is that the bile cannot pass into the small intestine as it should, so you wish to get at the lesion which is obstructing the passage of the bile from the liver into the intestine. More than this, you have to vary your treatment according to the particular part of the mucous membrane which is involved. In those cases in which there is gastro-duodenitis, when the stomach, common bile-duct, and duodenum are involved, you have, in the first place, to treat the stomach. The treatment is the same as in a simple case of gastritis. The patients are confined to a fluid diet, given in small quantities. You should allow them to take ice and ice-water as much as they like, even if they vomit it. You give such drugs as are ordinarily given in acute gastritis, such as oxalate of serum, bicarbonate of soda or potassium, hydrocyanic acid, calomel in small doses, for it is to act upon the stomach and not upon the bowels. In addition to these you may try counter-irritation over the epigastric region by means of blisters applied over the pyloric end of the stomach. In those cases where the stomach is not involved there is no gastritis to be treated. Counter-irritation, is, however, applied as before over the epigastrium, and in addition the patient is purged to a moderate extent by means of any one of the drugs which act pretty promptly, such as calomel, castor oil, or the sulphate of magnesia. In those cases in which only the bile-duct is involved, the treatment is about the same. When the stomach is involved it is also proper to empty the intestine, but before doing this it is customary to wait until the symptoms of gastritis have subsided, or, in other words, after the vomiting has ceased, when you can give a cathartic medicine with more certainty. When there is considerable fever and prostration the patients have to be confined to bed, when there is no fever they may be allowed to be up, and even go out of the house if the weather is tolerably pleasant. As a rule, there is no indication for the use of stimulants, even if the patients do seem somewhat prostrated. Sometimes, when the pain is a prominent feature, it is necessary to relieve it by opium, and the best way of doing this is by means of small hypodermic injections of morphine.

In those cases in which there is fatty infiltration of the liver, in addition to gastro-duodenitis, you treat the patients in the same way as those having gastro-duodenitis without a fatty liver, but you do so without expecting the same good results. You expect the disease will continue, and the patient finally die of the disease. Such patients are apt to be old drunkards, and if the pulse becomes very feeble they may require some stimulants.

ABSCESS OF THE LIVER.—*Anatomical Varieties.*—There are three anatomical varieties of abscess of the liver. In the first place, abscesses are formed in the liver as the result of inflammation of the bile-duct; in the second place, they are formed as the result of inflammation of the veins of the liver; in the third place, abscesses are formed in the liver as the result of the lodgment of the emboli in connection with pyæmia; and in addition, abscesses in the liver are developed idiopathically—without any known cause. These last cases are the ones we will consider at present.

IDIOPATHIC ABSCESSES OF THE LIVER.—*Pathology.*—In this form of abscess of the liver there is usually not more than one, the exception being that there may be two, three, or four abscesses of considerable size. Most of them are as large as a man's fist, and some are even larger than this. They may be situated in any one of the lobes of the liver, or in any part of a lobe; but they are found more frequently in the right lobe of the liver than in any other. When once formed, their natural tendency is to become larger, and as they do so they approach the surface of the liver, and when they have nearly reached this point there is usually a local peritonitis set up, in consequence of which a part of the liver becomes adherent to the adjacent soft parts, and when the abscess becomes still larger, and perforates the capsule of the liver, instead of the pus escaping into the abdominal cavity, it usually burrows into the soft parts which have become adherent to the liver. These abscesses do not very frequently perforate into the peritoneal cavity. They more frequently perforate into the pleural cavity, or the lung may become adherent to the diaphragm and the pus escape into the lung tissue, and be coughed up by the patient, or the pus may burrow through the abdominal wall, and finally escape externally, or the abscess may extend to the stomach, or to the pelvis of the right kidney, and the pus be discharged into one or other of these cavities. These abscesses of the liver, as a rule, do not look like ordinary abscesses. The pus which they contain is of a thin, unnatural character. It does not look like the pus found in connection with wounds. It is not a thick, yellow, healthy pus, but it is thin, and looks more like purulent serum. It is very apt to contain a considerable amount of blood, and on this account may appear of a red color. Very often it contains broken-down liver-tissue, and then it is rendered thicker and more grumous. The walls of the abscess are not smooth and covered with pyogenic membrane, but are ragged and formed of broken-down liver-tissue. The appearance of the pus impresses you with the idea that the process is quite as much necrotic as inflammatory; that it is not alone inflammation, but that the cavity in the liver is formed as much by the breaking down and death of the tissue as it is by the replacement of tissue by pus.

Occurrence.—These idiopathic abscesses of the liver are very much more common in tropical than in temperate countries; in tropical countries they are one of the ordinary diseases. In New York, the disease is not very uncommon. We see every year a number of cases of abscess of the liver, the patients not having been in a tropical region or even out of New York. Cases also occur in the suburbs of New York. The disease is endemic to this particular locality.

Etiology.—In a certain number of cases, there are, at the same time, the lesions of dysentery. Sometimes the symptoms of dysentery precede those due to the abscess, or they may occur at the same time. This concurrence of the two diseases has led to the supposition that some of the cases of abscess of the liver were due to the dysenteric process, and to carry out this idea, it has been supposed that pieces of necrotic matter in the intestine are carried into the branches of the portal vein and lodged in the liver, thus giving rise to the abscess. This is possible, but we have no real demonstration of it, and besides, a much larger number of cases of abscess of the liver occur without any lesion of the large intestine.

Symptoms.—The symptoms of abscess of the liver vary considerably in different cases, principally in regard to their definiteness. There are some cases which are very difficult to make out, because the symptoms are so indefinite. There are others in which characteristic local and constitutional symptoms are perfectly well marked, and in these the diagnosis is much easier. The symptoms which belong to this disease are rigors and a febrile movement, the latter being very often followed by sweating. The rigors occur not only at the commencement of the disease, when the pus is being formed, but they may occur from time to time for a period of several months, being followed by exacerbations of the febrile movement, which in turn is followed by sweating. Occasionally there are cases in which a febrile movement is present all the time, though it is not always of the same intensity throughout the entire day. In other cases, the febrile movement will be intermittent, there being hours and days when it is absent altogether. These intermissions sometimes occur as regularly as do those in intermittent fever. Pain is a regular symptom, and when it exists, it may be quite severe. It is usually described as a dull, steady, throbbing, deep-seated pain, and is referred to the region of the liver. Jaundice is not a regular symptom. It is possible for these patients to be jaundiced, but, as a rule they are not. Vomiting and diarrhœa are inconstant symptoms. If present, they differ as to their severity, the patient vomiting a good deal, or a little, or the diarrhœa being marked, or very slight, or none at all. Of course if the disease is complicated with dysentery you have the characteristic dysenteric passages added to the other symptoms. If there is local peritonitis, you may have the symptoms belonging to this, a local pain, increased febrile movement, and prostration. Occasionally, there is an effusion of serum into the peritoneal cavity—ascites. When the abscess ruptures into the pleuritic cavity there is added the symptoms of empyema to those of abscess of the liver. When it ruptures into the lung, the patients cough up the pus, and, in addition, will have symptoms referable to the lung which is perforated by the pus. The spleen, as a rule, is not enlarged. The liver, however, is generally enlarged and the particular way in which the enlargement occurs depends upon the position of the abscess. Besides all these, the general condition of the patient suffers. They lose flesh and strength and become more and more ill. They may not, at first, be confined to bed, but after a time they take to the bed and become more prostrated.

Duration.—The disease is commonly one of some duration. The bad cases may indeed terminate fatally at the end of two or three weeks, but, in the milder cases, the duration is much longer. The patients may live for several months, three, four, five, or six months, and although the disease is always serious, it is however, possible for them to get well.

Physical Signs.—The physical signs of abscess of the liver, depend upon the particular part in which the abscess is situated. If it is situated near the middle of the right lobe, the liver will be enlarged upward, toward the pleuritic cavity, and the abscess, in these cases, will usually rupture through the diaphragm into the pleuritic cavity, or into the lung. In these cases we may make out an enlargement of the liver behind and not in front. The enlargement does not take place downward, but upward, and this upward enlargement can usually be best appreciated behind. In a normal individual we should not get any dulness whatever behind, but when the liver is enlarged in persons who have abscess, the line of dulness will extend upward and you will get liver dulness, even though the organ is overlapped by the lung; you will get dulness low down upon the right side. This is the place where you are apt to find evidences of the enlargement of the liver in cases of abscess occurring in the upper part of the right lobe. In other cases the abscess may be situated in the right lobe anteriorly and lower down. If the abscess is in the lower part of the right lobe of the liver while it is still small, the only change that can be demonstrated will be that the liver extends below the free border of the ribs. As the abscess gets larger and larger, the liver becomes lower and lower in the abdominal cavity. It not only projects downward in this way, but the abscess will become large enough to push out the abdominal wall, and when it is of a large size, adhesions take place between the liver and abdominal wall and fluctuation can be felt as in any abscess. In other cases the abscess is situated somewhat higher in the right lobe of the liver. When thus located, the liver is only very slightly enlarged downward, but there is enlargement forward, so that the intercostal spaces are bulged out, very much like the bulging tumor formed by empyema, over the region of the liver and here again, if the tissues have become adherent to the liver, you can get fluctuation. When the abscess is in the left lobe of the liver, the organ is enlarged to the left and downward.

Diagnosis.—Of course, the readiness with which you make out the existence of one of these abscesses in any of the positions above mentioned, will depend upon its size. When small, you can only guess at its existence by the symptoms of the patient. When, however, the abscess reaches a considerable size, and you have fair reason to think that one exists, you are at liberty to plunge an aspirator into the liver. No injury of any consequence is caused to the liver by this procedure. You will observe, then, that cases of abscess of the liver vary both as to the definiteness of the constitutional symptoms and the physical signs. There are some cases in which both are so well marked that nobody can help making a diagnosis; there are other cases in which both are so obscure that a diagnosis is not made at all, and there are still other cases which present different degrees of both between these two extremes.

Treatment.—The treatment of abscess of the liver is directed to checking the formation of the abscess, to the removal of the pus and the supporting of the patient while the abscess is running its course. The preventive treatment we do not have an opportunity of carrying out. In tropical countries there is very often an opportunity for doing this. It is customary there to apply leeches and blisters, to give purgatives and alkalies, to give ipecac in twenty and thirty-grain doses, and chloride of ammonia in twenty-grain doses; but in New York we hardly ever have an opportunity of treating these cases. The patients do not present themselves for treatment until the abscess is fairly under way.

The indications for treatment when the abscess is formed are to empty it as soon as possible, and to support the general health of the patient as much as you can. In addition to this, if there is much pain it will be necessary to relieve it. If the abscess, at the time you see the patient, is already large and it is evident that adhesions have taken place between the liver and abdominal wall, you may open it by a free incision through the skin, and allow the pus to escape in this way. More frequently, however, we see these patients before such adhesions have taken place, and yet we feel confident of the existence of an abscess. Under such circumstances, we use an aspirator having a needle of large size. If a small needle is used it is apt to be plugged by fragments of necrotic liver tissue, which are drawn into it. We do not hesitate to plunge the needle of an aspirator into the liver, for very little danger is attached to the operation, nor do we hesitate to repeat this procedure several times if it is necessary. In this way we draw off as much pus as we can, and after a week or two repeat the aspiration. To support the general health of the patient we give quinine, iron, the mineral acids, and food in the shape of cream or cod-liver oil. If there is much pain we give opium in such doses as are necessary to bring it under control. But the most important part of the treatment is the evacuation of the abscess. This is a matter of the greatest consequence, and you should never wait too long before doing it. It is better to aspirate too soon and get no pus than to wait until the abscess is of large size, for the larger the abscess the worse the condition of the patient, and consequently the worse the prognosis.

LECTURE XLIII.

DISEASES OF THE LIVER—Continued.—CIRRHOSIS OF THE LIVER.

Cirrhosis of the Liver; Pathology; Associated or Secondary Lesions; Occurrence, Symptoms, Physical Signs, Rational Symptoms, Dropsy, Dyspepsia, Vomiting of Blood, Prognosis, Treatment.

Cirrhosis of the Liver.—*Gentlemen:* We begin to-day the consideration of cirrhosis of the liver, or chronic interstitial hepatitis.

Pathology.—This is an inflammation of the interstitial connective tissue which accompanies the veins and bile-ducts in their ramifications through the liver, and results in the formation of new connective tissue. The increase in the connective tissue stroma of the liver is accompanied, in most cases, with certain changes in the liver-cells. They usually become atrophied, or degenerated and atrophied, or they become the seat of fatty infiltration. In a certain number of cases there are also changes in the bile-ducts and veins of the liver. The small bile-ducts may become obliterated and the larger ones the seat of a chronic catarrhal inflammation, or, instead of this, there may be an actual production of new bile-ducts. The veins of the liver and the branches of the portal and hepatic veins may be obliterated by the presence of the new connective tissue. The inflammatory process is from the beginning a chronic one, there is no acute stage of cirrhosis of the liver. The new connective tissue which is produced is arranged somewhat differently in different cases. In the larger number it is so arranged as to surround or incapsulate the groups of acini of the liver. The surface of the liver will be thrown into lobules, which are surrounded by a zone of connective tissue, and when we examine the liver microscopically we find that these little lobules are a natural acinus surrounded by new connective tissue. Instead of surrounding groups of acini it may surround single acini. When this is the case, it will not have the ordinary appearance, but will look as if it were made up of a great many little granules, each of which corresponds to a little acinus, which is surrounded by a zone of new connective tissue. In still other cases, instead of the new tissue surrounding acini, it will follow the course of the veins and separate hepatic cells. In still other cases the connective tissue is arranged in the form of large bands, which run through the liver in different directions, and which divide it into large lobules. The gross appearance of the liver varies in different cases of cirrhosis, although the lesion is the same in all. This variation depends upon the particular way in which the new connective tissue is distributed, whether it divides the liver into large lobules or small lobules, or whether it surrounds single acini or grows diffusely between the individual liver-cells. There is also a difference in the size of cirrhotic livers. Some of them are of the same size and weight as normal; some are diminished in size and weight, weighing one or two pounds; others are increased, and weigh from five to ten pounds. In no case is the liver first enlarged and then diminished. In all cases it is first of natural size, and then, throughout the entire course of the dlsease, it may retain this size or it may gradually diminish in size as the case progresses, or gradually increase in size, but this difference in the size of the liver does not indicate any difference in the nature of the process, it being the same in all cases.

Associated or Secondary Lesions.—Cirrhosis of the liver is regularly associated with certain secondary changes in some of the other viscera of the body. These changes occur so regularly that we have to consider them as regular lesions of cirrhosis of the liver. They are all due to the same cause, namely, changes which take place in the branches of the portal and hepatic veins. A certain number of the branches of the portal and hepatic veins are pressed upon by the new connective tissue, and these are obliterated, and as a result, the blood can no longer pass from the liver as it should. The compression constantly increases the obstruction to the passage of the blood through the veins of the liver. In this way there is produced a chronic venous congestion of those viscera which naturally pour their blood into the portal vein, that is, the spleen, the stomach, and intestine become the seat of a chronic venous congestion. The result of the chronic venous congestion in the spleen is that it is increased in size and firmer than it should be. This increased size and hardness is due in some cases to an actual growth of new connective tissue. In the stomach, the chronic congestion involves principally the mucous membrane, which becomes the seat of a chronic catarrhal inflammation. It becomes thickened and is coated with an increased amount of mucus. In some cases there is a diffuse bleeding from the mucous membrane of the stomach in consequence of the chronic congestion. In the intestine we have changes similar to those in the stomach. The mucous membrane becomes the seat of a chronic catarrhal inflammation. It is thickened and coated with an increased amount of mucus, and may also bleed, although not as commonly as the mucous membrane of the stomach.

Besides these changes in the spleen, stomach, and intestine, there are transudations of serum from the vessels of the peritoneum, and as a result, we have accumulations of serum in the peritoneal cavity—ascites.

Occurrence.—Cirrhosis is regularly a disease of adult life ; it is indeed observed from time to time in children, but much more frequently in adults. It is observed most frequently in adults who have been habitually intemperate or who have been the subjects of constitutional syphilis, and if we get these two conditions combined, as we frequently do in the same person, we have the most favorable conditions for the production of the disease. We also have to admit the existence of cirrhosis of the liver without any known cause. This is especially the case when it occurs in children and old persons, and there are adults in whom we can get no history of syphilis or of the use of alcohol, and yet who are the subjects of this lesion.

Symptoms.—The evidences of cirrhosis of the liver are partly rational symptoms and partly physical signs.

Physical Signs.—The physical signs depend upon three conditions, the size of the liver, the size of the spleen, and the existence of ascites. In the first place, in regard to the size of the liver. If it is of normal size, it does not help us to exclude the existence of cirrhosis, and on the other hand, it does not help us to make a diagnosis. If the liver is enlarged we make it out very readily by palpation if there is no fluid in the peritoneal cavity ; but if there is a good deal of fluid in the peritoneal cavity, it will be very difficult to determine an enlargement of the liver, and it is sometimes impossible to make out whether the liver is smaller than normal or not. The pressure of the serum in the peritoneal cavity will not only protrude the abdominal wall forward, but press the liver upward into the concavity of the diaphragm and the liver will appear to be smaller. Again, beginning from above you find dulness over the upper edge of the abdominal cavity. but the dulness over the lower part of the thorax will be due to the presence of serum and not to the liver, and in some cases there is no difference between the dulness given by the liver and the dulness given by the serum in the peritoneal cavity. When this is the case, you cannot make out the size of the liver by percussion. Not infrequently there is a difference between the dulness given by the liver and that given by the fluid. This seems to be due to the presence of the intestine floating in the fluid. It is dulness of a tympanitic quality and quite frequently you can make out the lower edge of the liver in this way by the difference in the quality of the dulness. There are other cases which are a little more difficult to make out even than this. They are those in which the liver is of small size and there is a large amount of serum in the peritoneal cavity, but the serum instead of pressing the liver up against the diaphragm will get between it and the diaphragm so that you will have fluid above, then a small liver, and below, fluid again. The liver may be pressed down below the free border of the ribs, and s thus frequently mistaken for an enlarged liver. It can be felt through the abdominal wall in the abdominal cavity; but this is not because the liver is increased in size, but because it is pressed downward by the fluid between the diaphragm and liver. This condition is not of very frequent occurrence. When the liver is increased in size, it is usually pretty easy to make it out by palpation. You are able to feel the lower edge of it in the abdominal cavity. If, however, at the same time, the liver is enlarged and it is also coarsely nodular, you may be able to feel the nodules through the anterior abdominal wall. For this reason you may suppose that it is cancerous.

We make out the size of the spleen by percussion, and when cirrhosis of the liver is suspected you should never neglect to determine the size of the spleen, for this is always of use in making a diagnosis, especially in those cases where the liver is of a natural size, or where its size cannot be made out. In trying to determine the size of the spleen, have the patient lay down upon his right side, so that the fluid in the peritoneal cavity will gravitate to that side. You have then to remember the natural position of the spleen. The point you should look for is its anterior border. If you draw a line downward from the axilla until it cuts the tenth rib, this point will be at the anterior border of the spleen. The width of the spleen corresponds to the ninth, tenth, and eleventh ribs. You should be able to get the percussion dulness of the spleen as far back as a point nearly vertical to a line from the angle of the scapula. This varies more than the anterior limit on account of the position of the lung, for it may lap over a little so that you will not get dulness quite as far back as this. Remembering, then, where the spleen ought to be, you can percuss in different directions: first, from before backward until you get the anterior border, then percuss from above downward until you get the upper edge, and from before backward until you get the posterior edge. When the spleen is considerably enlarged this takes place downward and may be made out by palpation through the abdominal walls.

The ascites you make out by percussion, placing one hand upon one side of the abdomen, while with the other hand you tap the abdominal wall, and you appreciate a wave of fluid with the hand which is kept in position upon the abdomen. Besides this, by tapping the abdominal wall you get flatness below the level of the fluid.

Rational Symptoms.—Now, when you come to the rational symptoms, you find that these are of two kinds. They depend, in the first place, upon the interference with the digestion of the patient and upon changes in the nutrition due to these disturbances of digestion, or they depend upon the chronic venous congestion in the stomach, spleen, and intestine. We will consider first the symptoms due to the chronic venous congestion consequent upon changes in the veins of the liver: first, dropsy; second, dyspepsia, due to the chronic gastritis; and third, vomiting of blood.

Dropsy.—This begins regularly in the peritoneal cavity, and may remain confined to this. Sometimes it will begin at the same time in the peritoneal cavity, in the legs and in the feet. Again, there may be dropsy of the feet before there is dropsy of the peritoneal cavity. When this happens it indicates cirrhosis and chronic Bright's disease. The rule is that the fluid begins to make its appearance first in the peritoneal cavity. If the case lasts for a long time, and serum continues in the peritoneal cavity, after a while there is added general dropsy. There are accumulations of serum in the pleural cavity, pericardium, and a general œdema of the connective tissue throughout the whole body, and the patients become the subjects of general anasarca.

Dyspepsia.—The symptoms due to congestion of the stomach are the ordinary symptoms of gastric dyspepsia. The patients may suffer from nausea, with or without loss of appetite. They may have pain in the region of the stomach, or any of the symptoms of gastric dyspepsia.

Vomiting of Blood.—The vomiting of blood is not a very frequent symptom of cirrhosis of the liver, but it is observed from time to time. Sometimes it is vomited in small quantities; at other times it is vomited in very large quantities, so large as even to prove fatal. Such vomiting of blood is secondary to the condition of the liver.

Besides these we have other symptoms which are dependent upon the disturbance of the functions of the liver. These consist in the gradual loss of flesh and strength on the part of the patients; they become anæmic. Their mental faculties are affected; they are dull, stupid, drowsy, and are unable to attend to their ordinary business, and in the worst cases this gradual loss of nutrition may be sufficient to cause death. These two sets of symptoms are sometimes associated in the same patient; at other times not. That is, you may find in some patients the dropsy, disturbance of the stomach, gradual emaciation, gradual loss of strength; or you may find these two sets of symptoms disassociated; the patient may be very dropsical and his nutrition very good; he may continue to have a fair appetite and digest his food well. His muscular strength and nutrition will not be impaired. Less frequently the converse of this proposition will exist. There will be no dropsy whatever, the patient will gradually lose flesh and strength, become anæmic, and may even die simply from failure of nutrition.

Cases of cirrhosis of the liver are very apt indeed not to be made out, for it is customary in thinking of them to expect dropsy; yet there are cases in which there is no dropsy and the patients finally die from loss of nutrition. There is still one other symptom which does not belong regularly to this disease, but which occurs sometimes. It is always possible for a cirrhotic patient to become jaundiced, and when this occurs it depends upon the catarrhal inflammation of the larger bile-ducts.

The course of the disease is regularly slow, lasting for months, and usually for years. The lesion is developed gradually, as well as the symptom. It is indeed possible for the lesion to develop to a considerable extent without producing any symptoms whatever. It is common enough for us to find at an autopsy a moderate degree of cirrhosis of the liver when the patient during life has given no symptoms whatever of this condition. There is a considerable difference in different cases as to the rapidity of the development of the symptoms.

Prognosis.—The prognosis of this disease is always bad, its natural termination being in the death of the patient. The patients die from dropsy, loss of nutrition, or some complicating disorder, but the fatal termination may be put off for a considerable number of years. The patients who live longest are those who suffer from dropsy, and not apparently from any interference with the functions of the liver. They may live for many years, the dropsy disappearing for a certain length of time, and then reoccurring, but they finally succumb to the disease.

Treatment.—The treatment in these cases is confined to the treatment of the symptoms. We know at present of no means by which we can directly affect the chronic inflammation which is going on in the liver, but we can do a good deal for the relief of the different symptoms of which these patients complain. The dyspeptic symptoms depending upon the chronic gastritis can be treated in the same way as the ordinary symptoms of gastric dyspepsia. The dropsy is to be treated by endeavoring to remove the fluid from the body by means of the skin, kidneys, or intestines, or we can draw it off directly from the abdominal cavity. Diuretics act better in the case of patients with cirrhosis than in those suffering from kidney disease. Any of the ordinary diuretics may be used, such as iodide of potassium, infusion of digitalis, or the resin of copaiba, which will cause the patient to pass an increased amount of urine. If, however, the abdomen is very much distended with serum, so that the kidneys and the blood-vessels going to the kidneys are compressed, you will find that diuretics will not act. Under such circumstances you have to tap the abdomen by introducing a trochar through the abdominal wall and allowing the serum to flow out. When you have drawn off the serum in this way, you will very often find that the diuretics which would not act before will now act very well. There is some difference of opinion as to the best manner of aspirating the abdominal cavity. Some prefer to use the ordinary trochar and canula ; others prefer the aspirator. There is something to be said for and against each of these, and it is, after all, to a great extent a matter of choice, the main point being to get rid of the serum in the abdominal cavity, and without setting up either an acute or chronic peritonitis. When these patients are tapped often, as is sometimes necessary, on account of the repeated reaccumulation of the serum, there is, as a rule, a chronic peritonitis set up. There are very few cases of ascites in which you do not find after death a moderate amount of chronic peritonitis, but this does not lead to extensive adhesions, and the patients do not seem to be much the worse for it. Occasionally tapping of the peritoneal cavity is followed by an attack of an acute peritonitis, which frequently proves fatal. To mprove the general health of the patient, you give iron, quinine, and the mineral acids and fats in the shape of cream or cod-liver oil, and such other general measures as are beneficial to the general condition of the patient, and in this way life can be protracted for a number of years. You will always find that the worst cases are those in which there is no dropsy, but in which there is a gradual loss of nutrition. The best cases are those in which there is dropsy, but no impairment of the general health.

LECTURE XLIV.

DISEASES OF THE LIVER — CONTINUED. — FATTY LIVER.—PARENCHYMATOUS INFLAMMATION OF THE LIVER.

Fatty Liver; Pathology, Occurrence, Symptoms, Treatment.—Parenchymatous Inflammation of the Liver; Pathology, Occurrence.—Yellow Atrophy of the Liver, or Idiopathic Parenchymatous Inflammation of the Liver; Pathology, Etiology, Nature of the Disease, Symptoms, Duration, Prognosis, Treatment.

FATTY LIVER.—*Gentlemen:* We will go on to-day to the consideration of another morbid condition of the liver—fatty liver.

Pathology.—It is that condition in which the liver-cells are infiltrated with globules of fat. There is no change in the blood-vessels or stroma, and there may be no change in the bile-ducts, the essential change being in the hepatic cells. The fat is found in these in the shape of globules of different sizes. A single cell may contain several small fat-globules, or it may be so distended with fat that only a narrow rim of cell-tissue remains. The accumulation of fat usually begins in the periphery of each acinus next to the branches of the portal vein. The gross appearance of a liver which is the seat of fatty infiltration depends partly upon the extent to which it is infiltrated with fat-globules, and partly upon the amount of blood contained in the liver at the time of the patient's death. If the process of infiltration is not very far advanced, but there is a considerable amount of blood in the liver at the time of the patient's death, the liver may have a normal appearance. If, however, the fatty infiltration is farther advanced, you find it regularly increased in size, and such a liver may become very large indeed, so as to weigh from five to nine or ten pounds. The color of these fatty livers varies. They may be of a light yellow, or red mottled with yellow, or sometimes stained of a greenish color by the bile. The consistency of fatty livers seems to vary according to the character of the fat with which they are infiltrated. Some are firm and hard; some soft and flabby. The smaller bile-ducts, as a rule, are not changed, but the larger ones may be the seat of a catarrhal inflammation, and under such circumstances there may be an accumulation of bile in the bile-ducts throughout the liver, resulting in the staining of the liver with bile.

Occurrence.—This condition of the liver is found at all ages—in infants, young children, young people, adults, and old people. It is more common in females than in males, more common in persons leading sedentary lives, and in those who consume a good deal of some form of alcohol, than in others. It is common in ordinary phthisis.

Symptoms.—The symptoms of fatty liver are sometimes well marked, at other times they are very slight; they are usually in proportion to the extent of the lesion. When there is but a moderate amount of fatty infiltration, there may be no symptoms at all indicating this condition, but when the infiltration is marked, as a rule, there are symptoms, the most common of which are due to derangement of the functions of the liver, and they resemble those which I have already spoken of to you under the head of functional derangement of the liver. The patients do not suffer very much in their general nutrition; they remain fat and well nourished, but they are the subjects of uncomfortable feelings of different kinds, which are referred to different parts of the body. At times patients will complain of a loss of memory, an inability to attend to business, or concentrate their minds upon any one subject, or of an unnatural drowsiness or lassitude, and an indisposition to exertion. In other cases, the patients complain of pains and unnatural feelings referred to the thorax or abdomen; not infrequently the bowels are disturbed, there being diarrhœa from time to time. Although the patients remain fat and well nourished, they may become somewhat anæmic and unnaturally pale, and they may lose strength. The stomach may be disturbed, patients complaining of a loss of appetite, nausea, and sometimes vomiting, and, though rarely, the vomiting of a small amount of blood from time to time.

If the liver is much increased in size, you can make it out by physical examination. It is usually symmetrically enlarged, projecting down into the abdominal cavity, which can be determined without great difficulty.

When patients who have already a fatty liver become the subjects of gastro-duodenitis, they suffer from the symptoms I have described to you under the name of simple jaundice. You may remember, that in addition to the ordinary symptoms of jaundice accompanied with gastro-duodenitis, we have cerebral symptoms. These are common in fatty infiltration of the liver. Patients having fatty liver sometimes acquire acute gastro-duodenitis, and the combination of these two conditions is very apt to result in the death of the patient.

There is still a third set of cases in which the liver becomes very much enlarged. In these, the nutrition of the patients does suffer. They not only lose strength, but they also lose flesh. They become feeble and anæmic, and suffer from cerebral symptoms and from the uncomfortable feelings referred to the thorax or abdomen, of which we have already spoken; but of the above symptoms the most noticeable are the loss of flesh and strength. It is in these cases, also, that we are most likely to have catarrhal inflammation of the bile-ducts of the liver, and, with this, jaundice. The jaundice is, as a rule, not very marked. The patients become of a light yellow color, but they do not become very yellow, and they are apt to remain jaundiced for a long time.

Diagnosis.—The enlargement of the liver can very readily be made out upon physical examination. Examples of this last set of cases are not infrequently mistaken for cancer of the liver, though you might think at first that there could be no possibility of confounding two such opposite conditions. In fatty liver, where the enlargement is marked, the changes in the general condition of the patient are as marked as in cancer, and, as in that disease, the patients are jaundiced, anæmic, and of a cachectic appearance. Upon physical examination you find the liver filling a large part of the abdominal cavity, and it may be difficult to decide whether the patient is suffering from cancer or fatty infiltration. This is the worst form of fatty liver, the patients dying from lack of nutrition, in a feeble and emaciated condition.

Treatment.—The treatment of these patients varies with the extent of the lesion in the liver. If the lesion is not very much developed, and if the patients still retain a fair amount of health, although the functions of the liver are disturbed, a great deal can be done to improve their general condition by hygienic treatment: the treatment by means of drugs amounts to very little. These patients should be compelled to give up the use of alcoholic drinks altogether, and to take regulated exercise, at first of a moderate nature, and afterward more vigorous. They should not be allowed to eat fat in any shape. Their diet should be composed principally of meat and fruits. Under such treatment the symptoms gradually improve, and the patients return to a natural condition of health. In very bad cases, however, in which the liver is of a very large size, treatment does not seem to be of any avail whatever. These patients are too feeble to take any exercise, and you have simply to feed them as well as you can, and to make their lives as comfortable as possible, knowing that the fatal termination of the disease will not be put off for a very long time.

PARENCHYMATOUS INFLAMMATION OF THE LIVER.—By parenchymatous inflammation of the liver we mean that the inflammatory changes are confined to the liver-cells. The blood-vessels, the stroma, and the bile-ducts are not involved.

Pathology.—At the commencement of this process, the only change that we see in the liver-cells is that they are increased in size. After the inflammatory process has lasted a little longer, we see that the material of which the bodies of the liver-cells are composed is changed in its character. It is no longer as homogeneous as in the natural cells; it is becoming coarsely granular. As the process continues we find the production of coarse granules becomes more and more marked, and added to these are little drops of fat. When the cells are so much changed as this, we are no longer able to see their nuclei. The final result of the process is that the liver-cells break down, disintegrate, and then we find nothing but a mass of amorphous granules.

The change in the liver-cells is accompanied regularly by a disturbance of all the natural functions of the liver. The production of bile is diminished, the regular chemical change in the blood, as it passes through the liver, does not take place, and glycogen is not produced.

Occurrence.—This form of inflammation presents as a secondary and a primary lesion. As a secondary lesion it is very common indeed, but as a primary lesion it occurs less frequently. We find it as a secondary lesion in a considerable number of the more severe general diseases, as yellow fever, typhus and typhoid fevers, cerebro-spinal meningitis, pyæmia, acute general tuberculosis, cholera, the acute exanthemata, pneumonia, poisoning by phosphorus, arsenic, and antimony. Sometimes we find this condition of the liver not far advanced; at other times we find it advanced to a considerable extent. When it occurs as a secondary disease, we are not commonly able to make out any disturbance of the liver proper, for the symptoms due to this condition are marked by those belonging to the primary disease. For example, in yellow fever, although the changes in the liver are very marked, yet we do not find any liver-symptoms, owing to the fact that those of yellow fever are so marked as to conceal those due to the condition of the liver. The same is true of other general diseases complicated with parenchymatous inflammation of the liver.

IDIOPATHIC PARENCHYMATOUS HEPATITIS.—Idiopathic parenchymatous inflammation of the liver gives rise to very marked symptoms. This condition is known by several names. It is sometimes called, as we call it, by the name of acute parenchymatous hepatitis. Sometimes it is called acute yellow atrophy of the liver, or malignant jaundice. Perhaps the most common name is that of acute *yellow atrophy of the liver*.

Pathology.—The liver is usually diminished in size, and it is for this reason that the name of acute yellow atrophy has been given. Sometimes this diminution in size is so rapid, that you will be able to determine that it becomes smaller from day to day, and from hour to hour. In moderate cases, by the time the patient dies, the liver will weigh between one and two pounds, instead of three or four pounds, as the normal liver should. Although the rule is, that it is diminished in size, it is possible for patients to suffer from all the symptoms of parenchymatous hepatitis and die from this, and yet the liver will not be diminished in size or weight.

As a general thing, the liver is not only changed in size, but in consistency. It becomes soft, flabby, and brittle, so that it is easily torn. The blood-vessels are usually empty. The bile-ducts and gall-bladder are also empty, or there may be very little bile in the gall-bladder. When we examine the liver with a microscope we find the changes in the hepatic cells of which I have already spoken. Accompanying these changes in the liver, we most frequently find the spleen increased in size. We do not find any changes in the stomach or intestine. The kidneys are regularly the seat of marked lesions—of parenchymatous inflammation and changes in the cells lining the tubules of the kidneys. The same changes exist in the hepatic cells. There are also extravasations of blood into serous cavities as into the pleura, pericardium, and peritoneum; sometimes, though not as frequently, there are extravasations of blood into the lungs.

Etiology.—As to the causation of this disease we are very ignorant. It has been observed at all ages, in both males and females, although more cases occur in males. It has been observed in pregnant women, and also in connection with constitutional syphilis.

Nature of the Disease.—There is considerable difference of opinion as to the nature of the disease. Some believe that the lesion is primarily a local one, and that the symptoms are due to the inflammatory condition of the liver. Others, however, believe that it belongs to the class of general diseases, and that it is, like typhoid fever or cerebro-spinal meningitis, a general disease with a characteristic local lesion. It is still doubtful whether we are to look upon this condition as a simple local inflammation accompanied with characteristic symptoms, or a general disease with a characteristic local lesion.

Symptoms.—The invasion of the disease is sometimes acute; at other times slow. In the slow cases the patients at first complain of a moderate disturbance of the stomach, there being a little nausea, occasionally vomiting, and a little prostration. You might think the patients were simply suffering from ordinary catarrhal gastritis. They will be up and about. The stomach symptoms may last for several days, or they may even last for two or three weeks before the more marked symptoms of the disease develop. In other cases, however, the invasion of the disease is sudden. A patient who has previously been in good health, will be suddenly attacked with vomiting, prostration, and a febrile movement, and in a few hours will become jaundiced, and develop cerebral symptoms. Vomiting is a very constant symptom. It occurs at the commencement of the disease, both when it begins suddenly and gradually; but when the invasion is sudden the vomiting is much more marked and constant, than when it comes on slowly.

When the disease is fairly established, there are certain well-marked symptoms. The vomiting continues throughout its entire course. After having first vomited the food, the patients continue to vomit a greenish fluid, and sometimes a coffee-ground fluid in which there is decomposed blood. Jaundice always develops, and is apt to be well-marked. It usually occurs as soon as the other symptoms are fairly marked. In addition to the jaundice, there are the clay-colored stools. The urine contains bile pigment, such as is obtained in ordinary cases of obstructive jaundice. The pulse is full and rapid at first, and as death approaches, it becomes more rapid and feeble. Sometimes the jaundice does not make its appearance until six or seven days after the commencement of the disease, or after the patients have become fairly sick; and in exceptional cases, it occurs within twenty-four hours of the death of the patient.

A considerable number of patients complain of pain in the region of the liver. It is not severe, but the feeling is a dull and disagreeable one. As a rule, there is a febrile movement during the course of the disease, but this is exceedingly irregular. Sometimes it will be present at the commencement of the disease, and will diminish before the death of the patient; in other cases the fever will not come on until later. There are, however, cases in which the temperature at no time will be over 99° or 99.5°; not infrequently the temperature falls to 98° or 96°. These rises and falls in the temperature seem to have no reference to the progress of the disease. The disease steadily progresses while the thermometer indicates an irregular temperature. Sometimes the temperature is as high as 107° for twenty-four or forty-eight hours, but more frequently not over 104°. The pulse follows to a considerable extent the febrile movement. When the temperature is high at the commencement of the disease, the pulse will be rapid and full; when the disease has lasted for a long time, the pulse continues rapid, but more feeble, and toward the close of the disease the pulse becomes very rapid and feeble. During the middle period of the disease, if there is not much fever, the pulse may become comparatively slow.

The cerebral symptoms consist of headache, delirium, convulsions, and coma. These symptoms are nearly always present. The one which is least constant is convulsions. Delirium and coma occur in all cases; headache in most cases.

In a considerable number of cases we can make out the size of the liver from day to day, and sometimes even from hour to hour we may determine that it is becoming smaller and smaller. The liver dulness will disappear altogether, and the organ will become so small that the intestine rises into the cavity of the diaphragm and obscures the liver dulness entirely. Hence, in some of these cases you may not be able to determine the size of the liver.

The urine is always changed. In many cases it contains albumen and casts, the casts being of the granular and epithelial varieties. Blood may be present in a sufficient amount to make the urine of a distinct bloody color. Albumen may be present in a very considerable amount. We also find in the urine substances called leucine and tyrosine, which are not common in normal urine. The normal amount of urea and uric acid is diminished very considerably. The presence of leucine and tyrosine, and the diminution in the amount of urea, are regular characters of the urine. These are due to disturbances of the normal function of the liver. The presence of blood-casts in the urine is due to parenchymatous changes in the kidney. As a complicating lesion, there is a tendency to extravasation of blood in the blood-vessels, and in this way we have vomiting of blood. The blood being decomposed by gastric juice, presents as a coffee-ground fluid. There may be passages of blood from the bowels. There may be ecchymosis of the skin which will give the patient the appearance of one suffering from purpura.

Duration.—The duration of the disease varies considerably with the invasion. If the invasion is gradual, the whole length of the disease may last for three or four weeks. If it is sudden most patients die within a week, and in other cases the patients usually die within a week from the time the cerebral symptoms are fairly developed. There are exceptional cases that die within twelve or twenty-four hours after the invasion of the disease. Most cases run a course of about a week, or a week from the time the cerebral symptoms develop.

Prognosis.—The prognosis of the disease is very bad indeed. The great majority of patients affected by it die. There have been, however, cases of recovery reported, and I think I have seen one such; but in the cases of recovery there has always been some doubt as to the diagnosis, as to whether they were not cases of acute jaundice instead of acute yellow atrophy of the liver.

Treatment.—The jaundice is to be met in the ordinary way. The cerebral symptoms are treated with chloral and bromides. The feeble heart's action is to be treated with stimulants. In the case which I saw that recovered, the patient took bicarbonate of soda for a considerable length of time, and I would be disposed to try this in other cases; but, after all, there is very little benefit to be derived from any treatment.

LECTURE XLV.

DISEASES OF THE LIVER—CONTINUED.—CANCER OF THE LIVER.

Cancer of the Liver; Occurrence, Pathology, Diagnosis, Symptoms, Treatment.

CANCER OF THE LIVER.—*Occurrence.*—*Gentlemen:* To-day we will consider cancer of the liver. This occurs as a secondary lesion; especially is it secondary to cancer of the stomach. A considerable number of patients suffer from the symptoms of both cancer of the stomach and of the liver, or from symptoms due to the primary cancer, wherever it may occur, and cancer of the liver. Occasionally, however, we do meet with primary cancer of the liver, but when this is the case it is apt to begin in the gall-bladder and extend to the liver.

Pathology.—Whether the cancer be secondary or primary, it consists of a new growth in the liver, sometimes in the form of nodules, sometimes in the form of a diffuse infiltration. The liver under such circumstances is, as a rule, increased in size, and it may be to a very great extent, so that nearly the whole of the abdominal cavity will apparently be filled by it.

Diagnosis.—If the new growth is in the form of nodules, these are usually of sufficient size for you to be able to appreciate them through the abdominal wall, which is of great help in the diagnosis. When you are able to feel through the abdominal wall a large nodular liver, it can only be one of two things, either a cancerous or a large, cirrhotic liver. When, however, the enlargement is in the form of a diffuse infiltration, the liver is enlarged regularly, and it cannot be determined whether the enlargement is due to a cancerous growth or to an infiltrated waxy liver. There are exceptional cases, in which no such enlargment exists, and these are sufficiently common for the fact to be borne in mind in practice.

Like cancer of the stomach, cancer of the liver occurs in persons advanced in life—over forty years of age; very seldom in persons younger than this.

Symptoms.—The symptoms depend partly upon the changes in the size of the liver, partly upon the presence or absence of a primarily new growth in some other part of the body, and partly upon the existence of the disease in a malignant form in the liver. Of the changes in the size of the liver we have already spoken. The spleen is, as a rule, not enlarged. This helps us in making a diagnosis between cancer and cirrhosis of the liver. In cancer the spleen is not increased in size; in cirrhosis of the liver it is increased in size. In cancer of the liver there is usually no ascites, and this again is of service in distinguishing between cirrhosis and cancer of the liver, ascites occurring as a rule in cirrhosis; but to this we have exceptions, there being cases of cirrhosis of the liver in which there is no ascites, and cases of cancer in which ascites does exist. If there is no cancerous lesion in any other part of the body, and there exists a primary carcinoma of the liver, the symptoms are most commonly, at first, very indefinite. It is noticed that the patients lose flesh, strength, and appetite, that their color is changed—they are becoming more and more anæmic. Afterward the patients will complain of nausea. Sometimes this will only amount to a feeling of discomfort; at other times there will be actual pain referred to the epigastrium and liver regions. These pains, however, are subject to much variation, for they occur sometimes in one part of the body, sometimes in another. They may come on as in biliary colic. A patient who has suffered from biliary colic may suffer from pain due to cancer of the liver, and the pain will be of the same character as when it was due to biliary colic. As the disease progresses the symptoms become more marked. The patient loses not only his appetite, but he also suffers from nausea and occasional vomiting, and he may vomit a little blood from time to time. The patients become more emaciated and anæmic, until the anæmia reaches such a point that they become the subjects of dropsical effusion. In this way there may be effusions of serum into the subcutaneous tissue of the legs, in addition to its taking place in the serous cavities, and there may be even some œdema of the face. In these cases the dropsy seems to be due to the condition of the blood and not to any obstruction in the circulation. If the liver is enlarged, this may be determined by physical examination. Commonly, these are all the symptoms of which patients thus affected will complain, the most marked symptom being a gradual depreciation in the general health of the patient. If the liver remains small it will be very difficult to make a positive diagnosis. You may be almost certain that the patient is suffering from cancerous disease in some part of the body, but you cannot say which particular viscus is affected.

If the new growth in the liver is secondary to cancer of the stomach, you have the ordinary symptoms of cancer of the stomach, which are very little changed by the secondary condition in the liver, the cases running through the ordinary history of cancer of the stomach, the only difference being that the liver may be increased in size, and there may be added the symptoms due to this.

Treatment.—There is, of course, no special treatment for cancer of the liver. We simply endeavor to keep up the nutrition of the patient as well as we can and relieve the pain as fully as possible, and hence, to render life as tolerable as we can; but really the only practical point, especially in cases of primary cancer, is to make out the diagnosis, and this, in many cases, is very difficult indeed.

LECTURE XLVI.

DISEASES OF THE LIVER—CONCLUDED.—BILIARY CALCULI.—SUPPURATIVE INFLAMMATION OF THE PORTAL VEIN.

Biliary Calculi; Occurrence, Number, Size, Color, Composition, Symptoms, Treatment.—Biliary Colic; Treatment.—Impaction of Calculus in Cystic Duct; Treatment.—Impaction of Calculus in Common Bile-Duct.—Suppurative Inflammation of the Portal Vein; Pathology, Etiology, Symptoms, Treatment.

BILIARY CALCULI.—*Gentlemen:* Now we go on to the changes found in the biliary passages of the liver, and in the first place, we will consider the concretions found in these passages—biliary calculi, as they are called.

Occurrence, Number, Size, Color, Composition.—Biliary calculi are found in the gall-bladder, in the cystic and hepatic ducts and small biliary ducts of the liver, but they are most commonly situated in the gall-bladder. The number of these calculi varies enormously, there being at times only a single calculus in the gall-bladder; in other cases there have been observed seven or eight thousand calculi in one gall-bladder. Their size also varies. A single calculus may be large enough to nearly fill the gall-bladder, while others may not be larger than the head of a pin, and between these extremes we have all gradations in size. The color also varies. Most of them are of a dark greenish color, but some are of a white, translucent color, composed principally of cholesterin and bile-pigment. In exceptional cases calculi will have formed around some foreign body which has acted as a nucleus. Such calculi have been observed to have formed around a dead parasite, needles or fragments of needles, and they have been observed to have formed around the stones of some fruits. Sometimes these calculi, in addition to being principally composed of cholesterin and bile-pigment, have a moderate amount of the salts of lime (the carbonate or phosphate of lime) in their composition.

We do not often find biliary calculi in persons under thirty years of age; exceptionally, however, they are found in children. These calculi are more common in females than in males, in the proportion of about three to two. It is considered that they are more common in persons of sedentary habits and those in the habit of taking rich food and alcoholic drinks than in any others, but certainly there are many exceptions to this.

Symptoms.—The symptoms of biliary calculi vary with the position of the calculus and with its movability, or in other words, whether the calculus remains in one place or moves along the biliary ducts. In the first place, these calculi may remain in the gall-bladder, and under such circumstances, in some cases, they may give rise to no symptoms at all. It is not very infrequent to find at an autopsy one, two, or three calculi in the gall-bladder, the existence of which has given rise to no symptoms during life. In other cases they do give rise to symptoms, the most important of which is pain, referred either to the epigastric region or a little to the right of this, very nearly corresponding to the position of the gall-bladder. This pain is usually very severe, but it is not constant, although the calculi are present all the time. There may be intervals of months during which no pain will be experienced, then pain will come on and last for days or weeks, after which it may disappear. In some cases the invasion of the pain is so sudden and severe that we have to suppose that the calculus becomes engaged in the cystic duct, remains in the opening a short time, and then falls back into the gall-bladder. If the pain of which the patients complain is not accompained by jaundice, and is the only symptom which they have, we can hardly suppose that the calculus has passed through the gall-duct. Sometimes, however, the patients will suffer from several such attacks, and there finally comes a time when there will be jaundice, which indicates that the calculus has not only engaged in the cystic duct, but has actually passed through it into the common bile-duct and into the duodemum. In still other cases, these calculi remain in the gall-bladder, but they set up an inflammation of its wall. In some cases this inflammatory process is very severe. It may result in suppuration, and when this happens the gall-bladder will become distended with pus and its wall infiltrated with it; then there will be a perforation, the pus escaping into the peritoneal cavity or adhesions forming between it and the soft parts; the pus makes it way into the soft parts. In other cases, there will be a simple inflammation at some particular part of the wall of the gall-bladder, and this may go on to perforation, when the calculus will escape into the peritoneal cavity or into the surrounding soft parts.

Treatment.—When the calculi remain in the gall-bladder they give rise to attacks of pain, and these have to be treated. There seems to be only two ways in which this pain can be relieved, namely, either by the use of opium or causing the patient to vomit. The cases in which vomiting seem to afford relief are those in which we have reason to suppose that the calculus engages in the cystic duct.

The mechanical effort of vomiting probably causes this to fall back into the gall-bladder, and thus relief is obtained. Whether this theory is correct or not, you will find that in a certain number of cases relief will be obtained by exciting vomiting. For this purpose the simplest emetics and those which act most promptly are the best. In some patients the drinking of warm water will excite vomiting; at other times mustard in warm water will act very promptly. Some patients are in the habit of putting their finger down their throat to cause vomiting. The yellow sulphate of mercury in five-grain doses may be used. Any one of these means will cause vomiting and thus stop the attack of pain for a considerable length of time. In some cases, however, this plan will not succeed, and the only resort is then to the use of opium; there is no other drug sufficiently powerful. The amount of opium given should be regulated by its effect in controlling the pain. Enough should be given to keep the patient moderately comfortable, knowing that the attack will not last very long—sometimes only a few days, at other times two weeks, but not very often longer than this.

Biliary Colic.—In other cases the calculi, instead of remaining in the gall-bladder, pass through the cystic duct and from this into the common bile-duct and thence into the duodenum. When the calculus pursues this course, it produces an attack of what is ordinarily called biliary colic. The most prominent symptom is pain, which is very intense, and is chiefly referred to the free border of the liver, but, begining here, it may radiate into both hypochrondriac regions and into the back and shoulders. The pain is not continuous. The patients may suffer from it for a few minutes, or hours, and then get better for a time, or it may disappear altogether, after having lasted half an hour, or several hours, and afterward come on as severe as before. These intervals of pain seem to correspond to the way in which the calculus is passing through the cystic or common bile-duct. The calculus does not usually pass through uninterrupted. When for a time it ceases to move, the pain becomes less, but as it begins to move again the pain comes on, and hence it is that patients have repeated attacks of pain until the calculus has passed through the common duct into the duodenum. The period required for the passage of the calculus in this way through the whole length of the cystic and common bile-ducts varies considerably. In some patients the whole procedure will only occupy a few hours; in others the attack will last for days. Besides pain, these patients are very apt indeed to complain of nausea and vomiting, these symptoms being most severe while the pain is lasting. When the pain passes off, the nausea and vomiting may disappear for a time, and then come on again. The bowels are usually constipated during an attack. The abdomen may become somewhat tympanitic, so as to resemble the abdomen of patients suffering from peritonitis; or some patients may suffer from jaundice. This symptom, however, does not come on until after the pain and vomiting have lasted for some time. When it develops, it continues during the attack and may last for a few days after its cessation. In some cases the gall-bladder will be distended, and when this happens you can appreciate it by palpation. You can feel it as a tense, elastic tumor, situated in the abdominal cavity in the natural position of the gall-bladder. These are all the symptoms found in ordinary cases.

There are cases, however, in which the attack is more severe than this, and in which the patients not only suffer from the symptoms enumerated, but also from considerable prostration. The heart's action becomes unnaturally feeble, the vital powers of the patient depressed, and this may go on to actual syncope. The patient may faint during an attack, and it is even possible for them to die, there being at least three recorded cases of death during an attack of biliary colic.

Treatment.—While such an attack of biliary colic continues, the indication which exists is to relieve the pain. We do this either by means of opium, chloroform, or ether. We give opium in the form of hypodermic injections of morphine, both because in this form it acts more promptly than in any other, and affects the stomach less. In some cases you give opium in as large doses as you dare, and yet it does not relieve the pain, though the patients are almost narcotized. Under such circumstances we use chloroform or ether, the latter, of course, being the safer. If it is likely that you will have to keep up the administration of an anæsthetic for a long time, you first give the patient a moderate dose (from five to ten minims) of morphine hypodermically, and a few minutes after this you allow him to inhale enough chloroform (or ether) to bring him partly under its influence, and keep up its administration for fifteen minutes or half an hour. By this time the opium will have had a chance to produce its effect upon the system, and you can stop the use of the chloroform, the effects of the opium being now sufficient to relieve pain. This, I think, is really the preferable way in the cases in which opium alone will not relieve the pain. Besides the use of opium and chloroform, or ether, it is also customary to put these patients in a warm bath. This is a very old method of procedure, and it seems to give relief in some cases; at all events, it does no harm, and it is proper to put the patients in a warm bath during the continuance of the attack. If the attack lasts for several days, it is proper to give some purgative medicine. If the stomach is very irritable, you have to give a simple enema of soap and water, or warm water and ox-gall mixed, or an enema of castor-oil. If the stomach is not too irritable, you may give a dessertspoonful of the tincture of aloes and myrrh, or ten or twelve grains of calomel, merely for the purpose of emptying the bowels. After the attack has passed off you have to endeavor to prevent the occurrence of similar attacks, and you do this, in the first place, by regulating the diet of the patients. You cause them to abstain from fats. The diet should be principally composed of meats, fruits, and starches. The patients should take a sufficient amount of exercise, and, if possible, travel. You should give one or other of the alkalies, bicarbonate of soda or potash, or one of the Vichies, Carlsbad water being the best of the alkaline mineral waters.

Impaction of Calculus in Cystic Duct.—In a large number of cases the calculus does succeed in passing through the bile-ducts and finding its way into the intestine. Sometimes, however, this does not take place, and the calculus becomes fixed at some point in one or other of the bile-ducts. If it becomes impacted in the cystic duct the result is dilatation of the gall-bladder without jaundice. The bile can escape through the hepatic and common bile-ducts, just as before, so that no jaundice occurs, the only mechanical result being dilatation of the gall-bladder. When the calculus becomes impacted in the cystic duct the gall-bladder is at first distended with bile, but if the impaction has lasted for some little time there is added to the bile a considerable amount of mucus and serum, produced by the wall of the gall-bladder, and finally the dilated gall-bladder will be filled principally with mucus and serum. In other cases the calculus acts as a sort of valve. It will allow bile to pass through the liver into the cystic duct, but will not allow it to flow back into the gall-bladder, into the common duct and cystic duct again. When this happens, the gall-bladder becomes more and more distended with bile. The increase in size of the gall-bladder differs considerably in different cases. In a considerable number of cases it will form a tumor about as large as an orange, which preserves the ordinary regular shape of the gall-bladder. The general health of the patient does not usually suffer very much. He suffers principally from pain and discomfort referred to the region of the gall-bladder. He will continue up and about, and will attend to his business, but will complain of a sensation of nauseousness and pain in the region of the gall-bladder, and upon examination you will find a tense elastic tumor situated in the natural position of the gall-bladder. In other cases the distention of the gall-bladder reaches greater dimensions than these. It will become so much distended that it will contain half a pint of fluid, or even more than this. There are cases upon record of enormous distentions of the gall-bladder from this cause. The patients usually suffer to a considerable extent from disturbances of the stomach; they have nausea, and vomiting. They lose flesh and strength, and suffer from an irregular febrile movement. They become very much prostrated, and the bowels are sometimes constipated, the constipation alternating with diarrhœa. In this way they become worse and worse, until they finally die exhausted by the disease.

Treatment.—In these cases it seems proper to attempt some mechanical interference. It is evident that this is the only way that the patients can be relieved, and this fact renders mechanical interference justifiable. The simplest way is to tap the tumor with a trochar or aspirator. If it has become adherent to the abdominal wall it may be tapped, but if you are not certain that such adhesions have taken place, it is more prudent to use the aspirator and empty the gall-bladder in this way. Besides this, an attempt has been made to open the abdomen and get at the gall-bladder in the same way that you operate for ovarian cyst. This operation, however, has only been successful in one case, which was in England. In that case the abdomen was opened, the gall-bladder drawn out and the calculus taken from it; afterward the edges of the gall-bladder were stitched to the edges of the wound and the patient made a good recovery.

Impaction of Calculus in Common Bile-duct.—Instead of becoming impacted in the cyst-duct, the calculus may become impacted in the common bile-duct. When this happens, we do have jaundice, which is persistent and becomes more and more marked. The color of the skin becomes of a deeper and deeper yellow, until the attack comes to a close. Besides jaundice, the patients suffer from pain referred to the region of the liver. They suffer also from nausea and vomiting, from the gradual loss of flesh and strength, from rigors and a febrile movement. The liver, as a rule, becomes symmetrically enlarged downward into the abdominal cavity, so that its lower border may be determined by palpation. After the calculus has been impacted for some little time, the part of the common bile-duct above it, the hepatic duct, and various of the hepatic bile-ducts in the liver become distended, and very often inflamed. Sometimes this is only a catarrhal inflammation, and then you will have mucus added to the bile which distends the bile-ducts. In other cases the inflammation is suppurative and will extend from the branches of the bile-duct into the liver to the liver-tissue, and in this way you have abscesses of the liver formed—small abscesses surrounding inflamed bile-ducts. These are the cases in which rigors and a febrile movement are especially apt to occur.

The termination of these cases is always in the death of the patient. There is no means as yet known by which such a termination can be prevented. The duration of the disease may vary considerably—months or years, but during its continuance the patients will constantly lose flesh and strength and continue to be jaundiced, and finally die, either exhausted as a result of the trouble in the liver, or from some intercurrent disease.

SUPPURATIVE INFLAMMATION OF THE PORTAL VEIN.—Besides affections of the bile-ducts which we have now considered, we have to consider a single disease of the portal vein and its branches which pass into the liver.

Pathology.—The portal vein may be the seat of a suppurative inflammation which will involve the wall of the vein and result in its becoming thickened and infiltrated with pus, and the cavity of the vein will be filled with the puriform fluid or coagulated blood or a small thrombus. Such a suppurative inflammation involves, in the first place, the trunk of the portal vein before it enters the liver. It extends from this to its branches in the liver, and from the walls of the branches to the liver-tissue. In this way a very large part of the liver-tissue may be replaced by pus—abscesses scattered through the liver following the course of the branches of the portal vein.

Etiology.—Such an inflammation of the portal vein with subsequent inflammation of the liver-tissue results from a variety of causes. It may be due to a traumatic cause. There are cases recorded in which fish-bones have passed from the intestines into the portal vein and there set up a suppurative inflammation. There is one case recorded where a piece of wire did the same thing in an infant after a ligation of the umbilical cord, the inflammatory process travelling back through the umbilical vein to the portal vein, where an inflammation was set up. In patients suffering from perityphlitis the inflammation may involve the superior mesenteric vein, one of the branches of the portal system. Ulcers of the stomach and intestine will also give rise to inflammation of the branches of the portal vein, and afterward of the main trunk. In the same way abscesses of the spleen and suppurative inflammation of the bile-duct may give rise, first to an inflammation of the branches of the portal vein and afterward to the main trunk. Severe blows over the region of the liver may sometimes set up such an inflammation in the portal vein, and, besides all these cases, there are those in which there is an inflammation of the trunk of the portal vein without any discoverable cause.

Symptoms.—The symptoms of suppurative inflammation of the portal vein are usually pretty well marked. The patients regularly suffer from severe pain, which comes on at the commencement of the disease, and is referred either to the epigastrium or to the region of the liver, or, if the case is complicated by peritonitis, as it not infrequently is, the area of pain will be more extensive, and will involve a considerable part of the abdomen. The pain at first, however, is distinctly local, and referred either to the epigastrium or liver. There is not only pain, but tenderness, the tenderness following the area of pain. The liver is most commonly increased in size, it being most marked after the disease has lasted for some little time, and after the formation of abscesses in the liver. The liver is not only enlarged, but it is also, as a rule, tender. Patients complain if you press over the region of the liver. The spleen is also regularly enlarged, and, like the liver, is often susceptible to pressure. Jaundice is the rule in these cases; but the rule is not absolute. It is possible for a certain number of patients thus affected to have no jaundice at all during the whole course of the disease. The patients do not usually vomit; there may be a little nausea, but no regular vomiting; although they do not vomit the contents of the stomach, they may vomit blood, and in a considerable quantity. Diarrhœa is quite a common symptom, and patients may even pass blood through the intestines. There are usually rigors and a febrile movement, but the rigors occur only at the commencement of the disease, or they may be repeated during its course just as in an ordinary idiopathic abscess of the liver, and, as in the latter affection, the febrile movement comes on at intervals, sometimes regular, sometimes irregular, and then is followed by profuse perspiration. If there is complicating peritonitis, the abdomen will become tympanitic and tender. The patients rapidly emaciate, become prostrated, and pass into a typhoid condition, in which condition they commonly remain until they die, for the termination of the disease is regularly a fatal one.

The invasion of the disease is in most cases acute; sometimes, however, it is not so. The patients will simply have peculiar symptoms of discomfort referred to the region of the liver, a feeling of a moderate amount of prostration lasting for some days before the pronounced symptoms develop. The disease, as a rule, lasts for two weeks, dating from the time of the first rigor. There are patients who die at the end of a few days. There are other patients whose lives are prolonged for a much greater length of time; patients may live four, five, six, seven, or eight weeks before death takes place; but sooner or later they die as a result of the disease.

Treatment.—As regards treatment there is very little to be said. The best we can do is to keep the patient as comfortable as possible while the disease is running its course.

LECTURE XLVII.

DISEASES OF THE KIDNEYS.—CHRONIC CONGESTION OF THE KIDNEYS.

Anatomy of the Kidneys; Function, Examination of Urine.—Group of Diseases known under the Name of Bright's Disease.—Chronic Congestion of the Kidneys; Etiology, Pathology, Symptoms, Treatment.

Anatomy of the Kidneys. — *Gentlemen:* We go on to-day to consider some of the diseases of the kidneys. The parts of the normal kidney that we have to remember in connection with pathological processes are, in the first place, the *capsule* of the kidney. In the normal kidney the entire organ is invested by a capsule of connective tissue, which is very loosely applied to the surface of the kidney, and is attached to it almost altogether by blood-vessels which run from it into the kidney tissue, so that, when we strip off the capsule, we leave the surface of the kidney perfectly smooth, except where the blood-vessels pass from it into the kidney proper. When you examine a kidney after death, and do not find the condition described to exist, when you find that capsule is adherent, and that when it is stripped off parts of the kidney come with it, you may be sure that it is not normal; that it is diseased.

Then we have the *pelvis* and *calices* of the kidneys, which are lined with perfectly smooth mucous membrane. The membrane should not be covered by any fluid whatever. If we find that the mucous membrane of the pelvis of the kidney has lost its smoothness, is thickened, congested, and covered with a purulent or muco-purulent fluid, we know that it is no longer in a natural condition.

When we come to the substance of the kidney proper we find that it is composed of a great number of *tubules, blood-vessels, Malpighian bodies,* and *stroma* which holds these together. The general course of the tubules through the kidney you will learn in your books of anatomy. The points we have to remember are that there exist what are called *straight* and *convoluted* tubes, and that the *epithelium* which lines these is of a different character in each instance. Besides this, we find that the convoluted tubes are more apt to become diseased than are the straight tubes. The straight tubes are lined with polygonal cells, the bodies of which are pale and transparent, and each contains a single nucleus. The cells lining the convoluted tubes do not have as distinct an outline. They look like a continuous layer of granular matter, in which are embedded nuclei at regular intervals. The bodies of the cells, instead of being transparent and homogeneous, as in the straight tubes, are somewhat granular.

The Malpighian bodies are composed of a capsule in which there is a tuft of capillary vessels. The capsule of the Malpighian bodies is simply a globular expansion of the convoluted tubes. It is lined by large, flat cells ; but when seen in section they look like flat polygonal cells. The tufts are continuous with the artery which pierces the Malpighian bodies and breaks up into a network of capillary vessels, which again becomes continuous with a small vein that emerges from the capsule. The surface of the capsule is again covered with little polygonal cells, which are normally scattered over the surface of the capillary vessels that make up the tuft. Of all the blood-vessels of the kidney the arteries are the least numerous. Most of the arteries terminate in the tufts of the Malpighian bodies. The venous system is very large indeed. Besides large veins we have an abundant capillary plexus, which runs between the tubules. These veins form the capillary plexus, which ramify both in the cortical and pyramidal portions of the kidney. The stroma of the kidney is composed of ordinary connective tissue, which, in the pyramidal portion, is tolerably abundant, but in the cortical portion is scanty. In some places in this latter situation the walls of the blood-vessels seem to constitute the only stroma which exists. There is, however, a considerable number of normal adult kidneys with a little stroma between the tubules, so that we look on this as a normal condition. The same thing is true of the liver, so that the mere finding of a little connective tissue in the kidney is not sufficient reason for calling it diseased.

Function.—The normal function of the kidneys is, as you know, to excrete a certain fluid called urine, which consists principally of water holding in solution a number of substances, which are for the most part prepared in the liver for excretion by the kidneys. The functions of the liver and kidneys are very closely related. Most of the solid substances excreted by the kidneys are, first and most important, urea and uric acid. About five hundred grains of urea are excreted daily by healthy individuals. Most persons normally have a little albumen in their urine, and a little sugar ; but these are present in such small amount that they are not usually found. There are many persons, who, after eating a considerable amount of sweet, will, in a few hours, pass urine which contains sugar ; hence, small quantities of albumen and sugar must be considered as normal constituents of the urine.

Examination of Urine.—When we examine a specimen of urine to determine whether the kidneys are healthy, we notice, in the first place, the quantity of urine passed during the first twenty-four hours.

This is always a matter of great importance, for persistent variation from the normal amount is in some cases the only evidence we have of well-marked disease of the kidney. Healthy adults ought to pass from forty to fifty ounces of urine during the twenty-four hours, but there are a number of healthy persons who habitually pass no more than thirty ounces, and some who even pass no more than twenty-four or twenty-five, and yet this amount will be consistent with health. If a patient, for a considerable length of time, passes a less amount than thirty ounces, or more than fifty, during the twenty-four hours, it may be sufficient evidence to suspect the kidney. The specific gravity of the urine should be noted, the normal average being 1,025; but here, again, we have variation within the limits of health from 1,020 to 1,030, but if you find that, for a considerable length of time, the patient passes urine of a specific gravity less than 1,020 or more than 1,030, it is a very good reason for suspecting disease of the kidneys. In addition, we test the urine in the ordinary way for albumen and sugar, but we must remember that these substances, when present in small amounts, do not necessarily indicate disease of the kidney; but if they occur in a considerable quantity, or if, besides their presence, you have other indications of kidney disease, their presence is indicative of disease of the kidneys. We have also to examine the sediment of the urine microscopically. In certain diseases of the kidneys we find in this casts of the so-called hyaline, granular, nucleated, epithelial, and blood varieties. The way in which these are formed we will consider in connection with the diseases of the kidneys in which they occur. There are also found epithelial cells from the bladder, ureters, and tubules of the kidneys. There is a very strong disposition on the part of some who examine urine microscopically to find in it renal epithelium, but in reality most of the epithelium that is so called is from the bladder; renal epithelial cells are not very common in urine. We also find in urine pus and blood and various crystals. Of the crystals, the only ones of much clinical importance are those of the oxalate of lime, phosphate of lime, and the urates in the form of crystals or in an amorphous condition. These, when present in excess, are of importance as indicating, not disease of the kidney, but disturbances of the digestion.

Kidney Diseases to be Considered.—The diseases of the kidney which we have to consider are, in the first place, the group which are included under the name of Bright's disease; in the second place, suppurative or tubercular nephritis; third, what is called cystic kidney; fourth, new growths of the kidney; fifth, renal calculi; and in the sixth place, inflammations of the capsule of the kidney and the connective tissue immediately surrounding it.

Group of Diseases known under the Name of Bright's Disease.—The term Bright's disease has become rather a popular one than a medical term. There would, perhaps, be a certain advantage if we could get rid of it altogether, but the name is so firmly fixed that this is not likely to take place; hence, the best way is to accept the term and try to use it in a rational way, and the only manner of doing this is by using it, not as expressing any one anatomical condition of the kidneys, but as expressing a group of diseases of the kidneys, which are characterized by certain changes in the urine—by the presence of *casts* and *albumen* in the urine. Those which make up this group of kidney diseases are, in the first place, chronic congestion of the kidneys; second, acute parenchymatous nephritis; third, chronic parenchymatous nephritis; fourth, acute diffuse nephritis; and fifth, chronic diffuse nephritis. All of these conditions are different, and yet we have the common symptom of albumen and casts in the urine, and for this reason we include all of these different diseases under the name of Bright's disease of the kidneys.

CHRONIC CONGESTION OF THE KIDNEYS.—*Etiology.*—This is not a special disease of the kidneys, but it is a condition which occurs in the kidneys in common with a similar condition in other viscera, and it is always due to a mechanical cause, namely, some obstruction to the general venous circulation of the body. The most common cause which effects such an impediment to the circulation of blood throughout the body is well-marked valvular disease of the heart or dilatation of its cavities. Either of these conditions will constitute a mechanical impediment to the venous circulation and certain of the viscera will suffer in consequence.

Besides this, we find sometimes that pericarditis with extensive effusion of serum will produce the same result. Emphysema of the lungs is not infrequently accompanied with congestion of the kidneys. Aneurism of the aorta, tumors of the thoracic and abdominal cavities may all act in the same way. We also find, occasionally, that pregnancy will produce chronic congestion of the kidneys. The viscera which most frequently suffer from chronic congestion are the lungs, liver, spleen, stomach, small intestine, kidneys, and less frequently the pia mater covering the brain. In all these different parts of the body we find essentially the same changes produced by some impediment to the free circulation of the venous blood. We find that the particular viscus which is affected constantly contains more venous blood than it ought, and as a result of this it becomes a little larger than normal and consequently a little firmer or harder than usually. After such a chronic congestion has lasted for a certain length of time there is added a chronic inflammatory process which involves either the cells of the particular viscus or the stroma, or both. Besides this, we find that the veins may become larger than normal, all of which changes are due to a mechanical impediment to the circulation.

Pathology.—In the kidneys we find, as a result of chronic congestion, if it has not existed for too long a time, simple enlargement of the organ, and increased redness, and this is all. The surface of the kidney will be perfectly smooth and the capsules will slip off as in health. When you examine the kidney you will find the tubes and Malpighian bodies structurally unaltered. If you examine the kidneys of a patient in whom congestion has existed for a long time, or one in whom the congestion has been more marked, the changes will be better defined. The changes will be confined to the convoluted tubes and other tubes, and these will be swollen and granular. In addition to the changes in the tubes, you will find a new growth in the connective tissue of the stroma; there will be added interstitial nephritis resulting in the production of new tissue, especially in the cortical portion of the kidneys. The blood-vessels of the kidneys usually do not indicate any change. These changes in the condition of the kidneys are associated during the life of the patient with certain changes in the condition of the urine and with certain general symptoms.

In the first place, if the kidney is simply the seat of chronic congestion, the epithelium of the tubules remains normal as well as the stroma. In these cases the urine is diminished in amount and its color deepened. Its specific gravity is higher than normal, and in addition to this it may contain a little albumen or none at all; it does not contain blood, although you might expect to find it.

If there is added to the chronic congestion changes in the epithelial cells of the tubes, the diminution of the quantity of urine will be more marked, and may go on to complete suppression for twenty-four, thirty-six, or forty-eight hours, or even longer than this. The specific gravity will remain about normal, or it may be a little increased. The urine may contain a larger amount of albumen, and in addition to this will contain casts either of the hyaline, granular, or nucleated varieties.

If, besides this, there are added changes in the stroma of the kidneys, the condition of the urine will be about the same as if the changes were confined to the epithelium, for there is in the same way a marked diminution in the amount of urine, and there may be complete suppression of it. Albumen may be present in a considerable amount, as well as hyaline, granular, and nucleated casts.

Symptoms.—When there is simply chronic congestion of the kidney without structural change, it does not usually produce any change in the general health of the patient. He will go on to suffer from organic heart disease, but the kidneys will perform their work well enough for all purposes, and no renal symptoms will develop. But, if the kidneys become the seat of structural changes in the epithelial cells and stroma, the patient will suffer from renal symptoms in addition to those due to the heart disease or other trouble from which he is suffering. These will be the same as the renal symptoms belonging to parenchymatous nephritis or chronic diffuse nephritis, so that it is not necessary to consider them here.

Treatment.—The indications for treatment in patients suffering from chronic congestion of the kidneys is to improve the general condition of the venous circulation. We do not attempt to do anything for the kidneys; but, although the cause of the obstruction to the venous circulation is mechanical and due to some structural change in one of the valves of the heart, yet we find that the amount of congestion in the kidney varies with the way in which the muscular tissue of the heart acts; when the heart acts well, the venous congestion is much less marked than when the heart acts too forcibly, too weakly, or too irregularly. We are able to act upon the heart's muscular tissue, but we can do nothing for the condition of the valves. If the heart beats habitually too feebly we can increase the force of the heart's action: we can increase the power of the muscular tissue by means of digitalis. If the heart beats irregularly, we can cause it to beat more regularly by the inhalation of nitrate of amyl or the use of iodide of potassium; if it beats too forcibly, we diminish this by the use of iodide of potassium, aconite, and other drugs. We find in addition that there is a certain relationship between the way in which the heart acts and the amount of blood contained in the arteries. If the arteries are habitually too full of blood, or there exists what is called a too high arterial tension, the result of this is to increase the amount of venous congestion. Here again, we find that we can do something to improve the condition of the patient, there being certain drugs which have the effect of diminishing increased arterial tension.

LECTURE XLVIII.

DISEASES OF THE KIDNEYS—CONTINUED.—ACUTE PARENCHYMATOUS NEPHRITIS.

Acute Parenchymatous Nephritis; Pathology, Occurrence; As Complicating Pneumonia, Typhus and Typhoid Fever; As Complicating Cholera, Yellow Fever, and Acute Yellow Atrophy of the Liver; As Complicating Scarlet Fever, Diphtheria, Peritonitis, and Poisoning by Phosphorus and Arsenic.—Acute Idiopathic Parenchymatous Nephritis; Changes in the Urine, Dropsy, Cerebral Symptoms, Duration, Prognosis, Treatment.

ACUTE PARENCHYMATOUS NEPHRITIS. — *Gentlemen :* The next condition of the kidney which we have to consider is *parenchymatous nephritis,* and in the first place we will speak of acute parenchymatous nephritis. This condition of the kidney again belongs to the general group of diseases, known under the name of Bright's disease, and is characterized by the presence of albumen and casts in the urine.

Pathology.—In acute parenchymatous nephritis the gross appearances of the kidney vary with the extent to which the lesion is developed; if it is only slightly developed, the appearance of the kidney will be very little changed, the capsule will not be adherent, the surface of the kidney will be smooth, and its size will not be changed; the only perceptible change will be that the cortical portion is a little whiter than that of a healthy kidney; if, however, the lesion is farther advanced, the kidney is increased in size, and this may be to a considerable extent, so that the two will weigh twelve, fourteen, or sixteen ounces, instead of nine or ten ounces, their normal weight. The capsule will not yet be adherent, and the surface will be smooth, but the change in the cortical portion will be much more marked, the color will be decidedly whiter than normal, and even the whole cortical portion may be almost of a uniform white color. The pyramids remain of the natural red color, although they may seem more intensely red by contrast with the whiter portion. When we come to examine these kidneys with the microscope we find that the lesion is confined to the epithelium of the tubes and the epithelium which lines the capsules of the Malpighian bodies. The stroma is not involved and the blood-vessels of the kidney are unchanged.

If the inflammatory process is not very intense, the changes will be confined to the convoluted tubes, and to the capsules of the Malpighian bodies: the epithelium of the straight tubes will not be involved. In these slighter forms of the inflammatory process the changes will not be very marked; they will consist simply in a swelling of the epithelial cells, and in a change in the substances of the cell-bodies, so that these become more opaque and coarsely granular than in health. If, however, the inflammatory process is more intense, we find, not only the convoluted tubes involved, but also the straight tubes, both of the cortical and pyramidal portions of the kidneys. The swelling of the epithelial cells is more marked; the granular matter is coarser, thicker, and more dense, so that the cells are rendered more opaque. Besides this; a certain number of cells fall off from the basement substance of the tubes, and are found in their cavities. Other cells disintegrate and break down, and the debris is found in the cavity of the tubes. Besides this, we find cast matter in the tubes of the pyramids and cortical portions, and this may be hyaline, or mucus with broken down and degenerated epithelial cells.

Occurrence.—Acute parenchymatous nephritis occurs both as a secondary and primary, or idiopathic disease. As a secondary disease it is very common indeed; as an idiopathic disease it is less common. The diseases with which it occurs most frequently as a secondary condition, are pneumonia, typhus and typhoid fever, cholera, yellow fever, acute yellow atrophy of the liver, scarlet fever, diphtheria, pyæmia, peritonitis, and poisoning by phosphorus and arsenic.

As Complicating Pneumonia, Typhus and Typhoid Fever.—The lesion, however, is usually developed in different degrees of intensity. in these different diseases. With pneumonia, typhus and typhoid fever the lesion of the kidney is usually not very pronounced, so that the complication is not a serious one, the patients not being made very much worse by it. As a rule, there are no renal symptoms, but we find changes in the urine. It contains albumen and casts, and is diminished in quantity.

As Complicating Cholera, Yellow Fever, Acute Yellow Atrophy of the Liver.—When, however, the parenchymatous nephritis occurs as a complication of cholera or yellow fever, or acute yellow atrophy of the liver, it is of a much more serious nature. The changes in the kidneys are much more marked, the inflammation being usually intense and well defined, and as a result of this the changes in the urine are much more decided. The patients have renal symptoms and the urine is very much diminished in quantity and may be completely suppressed for some length of time. The general renal symptoms which are especially apt to occur are the cerebral symptoms; the patients become delirious and stupid, and may have convulsions and die in a semi-comatose or a comatose condition. These symptoms seem to depend not upon the primary diseases, but upon the parenchymatous nephritis which occurs as a complication of them.

As Complicating Scarlet Fever, Diphtheria, Peritonitis-Poisoning by Phosphorus and Arsenic.—When it occurrs as a complication of scarlet fever, diphtheria, peritonitis, or of poisoning by phosphorus and arsenic, the changes in the kidneys are usually quite well marked, but are not as intense as in yellow fever or cholera ; they are more intense than in pneumonia, typhus and typhoid fevers. The changes in the urine and the renal symptoms are often fairly developed. The urine may be suppressed or diminished. It contains casts of the hyaline, granular, and nucleated varieties. The patients may become delirious and stupid. They do not often have convulsions, although this is possible. The stupor may become so marked that the patients pass into a condition of semi-coma—not usually into a condition of complete coma.

ACUTE IDIOPATHIC PARENCHYMATOUS NEPHRITIS.—When parenchymatous nephritis occurs as an idiopathic disease, sometimes patients will give a history of having been exposed to cold, or they will ascribe their condition to something in their habits of life, but in the larger number of cases we are not able to make out the exciting cause of the inflammation of the kidneys.

Changes in the Urine.—When it occurs as an idiopathic condition we find, in the first place, changes in the urine ; it is diminished in quantity, and this diminution in quantity may be very marked indeed. Patients may only pass a few ounces of urine during the twenty-four hours ; in other cases it will be completely suppressed for a considerable length of time. The specific gravity of the urine will remain unchanged, or it will be slightly increased, or very slightly diminished. The specific gravity does not change very much from that of normal urine. The urine commonly contains albumen, which may be present only in small amounts, but which is more frequently present in very considerable quantities, so that when you test for it by heat and nitric acid, you will find fifty, sixty, seventy, or eighty per cent. of albumen. Casts are commonly present in the urine (hyaline nucleated and granular casts), but, as a rule, they are not numerous for some reason or other. I do not know whether casts are very much more constant and numerous in parenchymatous nephritis occurring secondarily than when it occurs primarily. In idiopathic cases casts are usually present, but in very small numbers. You may examine specimen after specimen without finding any whatever.

Dropsy.—Besides these changes in the urine, we also have changes in the general condition of the patient. One of the most marked of these is the development of dropsy, which, as a rule, makes its appearance in the subcutaneous tissue of the legs and feet. When it has once commenced, it is apt to increase pretty rapidly, so that the legs become a good deal swollen ; afterward the dropsy extends to other parts of the body, and we have in addition the scrotum, abdomen, back, and thorax infiltrated. Serum accumulates in the peritoneal, pleural, and pericardial cavities. In a very considerable number of cases of idiopathic parenchymatous nephritis, the dropsy becomes a very marked feature; and here again there is a difference between idiopathic and secondary cases. In the secondary cases we do not, as a rule, have any dropsy. The secondary cases in which dropsy occurs are principally those arising in connection with scarlet fever; but with pneumonia, typhus and typhoid fever, cholera, acute yellow atrophy of the liver, phosphorus and arsenic poisoning we do not have dropsy.

Cerebral Symptoms.—In other cases, instead of dropsy being developed, there are cerebral symptoms; the patients become unnaturally drowsy; the drowsiness alternates with an unnatural restlessness, and even with delirium. These patients may also have convulsions. The drowsiness may change into stupor, or into a condition approaching coma.

In those cases in which there are cerebral symptoms, there are also very often disturbances of the stomach—the patients lose their appetite, are troubled with vomiting, which may be of a very persistent and distressing character. In still other cases, we have both these sets of symptoms—both dropsy, and cerebral symptoms, and disturbances of the stomach. Frequently these sets of symptoms are not associated in the same person.

Duration.—The duration of the disease is usually for a number of weeks, and not infrequently for several months. In very bad cases patients may only live for a few days; this is especially true when the cerebral symptoms are marked. The patients may die comatose at the end of a few days. But the cases in which dropsy is a prominent feature usually continue to live for a number of weeks and months, and gradually get better.

Prognosis.—The prognosis of acute idiopathic parenchymatous nephritis is tolerably good. A considerable number of the patients get well completely. The kidneys seem to return to the natural condition and perform their function as well as ever. But a certain number of the patients die early in the disease, with cerebral symptoms; others die after two or three months; and still others go on to develop chronic diffuse nephritis, and suffer from the symptoms of this condition; so that there is, at least, one form of Bright's disease in which the prognosis is comparatively good. Using the term Bright's disease, in the sense we have described, you can say that patients may recover completely from Bright's disease.

Treatment.—In the first place, as to the treatment of the very common cases—those in which dropsy is the only prominent symptom, and the general condition of the patient is pretty good, although the urine is diminished in amount, and contains a large quantity of albumen and a few casts.

The two symptoms which seem especially to need treatment are the diminution in the amount of urine and the dropsy. It is not wise to attempt to fulfil either of these two indications during the first two weeks of the disease while the dropsy is constantly increasing and the urine less in amount, for you will find the urine will remain the same in quantity and the dropsy will keep on increasing, whether you use diuretics, cathartics, or sweating, so that you disorder the patients' stomach and render their general condition worse without affording any relief. During the first few weeks (two or three, sometimes four, five, or six weeks) of an attack of idiopathic parenchymatous nephritis patients should be put upon a milk diet, and the progress of the dropsy watched. When the dropsy seems to have become stationary, and when the condition of the urine is apparently stationary, patients passing, as they usually do at this time, from fifteen to twenty ounces of urine containing a large amount of albumen, you can begin to interfere with advantage. If there is a good deal of dropsy, and the patient is not in a hospital, you will very often do best by attempting to get rid of the dropsy before increasing the quantity of urine. To do this you can take a pin and puncture the skin of the legs, scrotum, and abdomen. As soon as you have got rid of some of the dropsy in this way you will endeavor to act upon the kidneys. For this purpose you will find sometimes one, sometimes another of the diuretics will answer the purpose best, the acetate and iodide of potassium, infusions of digitalis, the bitartrate of potash, the pill composed of digitalis and squills, and the bichloride of mercury. These are all reliable diuretics, sometimes one answering best, sometimes another. If the diuretics begin to act, as they will do if the case is likely to prove favorable, the patients will begin to pass a great amount of urine—they will pass forty or fifty, or even sixty or seventy ounces a day. You do not usually care to have them pass more than seventy ounces of urine per day, and when they have reached this point, you can reduce the amount of the diuretic, but you should continue to administer it in quantities sufficient to keep the urine at this amount. If you do this, the dropsy will continue to rapidly disappear, and by the end of a couple of weeks the patient who has been in a condition of extreme dropsy will have very little, and as the urine increases in amount the dropsy and albumen diminish. When the case has reached this point you can allow the patient to gradually resort to his ordinary diet, and if it is during the warm weather to take more and more exercise, so that he can inhale the fresh air; if it is in the cold season he should be sent to a warm climate and remain there until entirely well. If the dropsy has disappeared and the patient is apparently entirely well, the urine will still contain a little albumen. It is evident that it requires a considerable lapse of time for the kidneys to get into a natural condition. The patient feels well enough and thinks that he is well, but albumen is present in his urine, and so long as this is the case, he should remain in a warm climate and not return to his regular business or occupation. In a certain number of cases convalescence will be accompanied with anæmia; the patients will become unnaturally white. When this happens, it is proper to give iron and inhalations of oxygen, and in this way improve the condition of the blood. It takes usually a year for the patients to return to a condition of complete health, although they may consider themselves nearly or entirely well at the end of three or four months.

In those cases in which cerebral symptoms occur, instead of dropsy, the indication for treatment is somewhat different. In these cases you cannot well afford to wait; the symptoms are too marked, they are two threatening; it does not seem safe to let the patient alone and trust to the natural progress of the disease. Hence we use the so-called eliminative means of treatment; we sweat the patient, act upon the bowels by purgative medicines and upon the kidneys by diuretics, though the administration of diuretics is not usually successful. Pilocarpin and jaborandi are given. You give small doses of the muriate of pilocarpin hypodermically, from one-tenth to one-twelfth of a grain twice a day, which will increase the perspiration and salivation and act upon the bowels.

The prognosis in these cases is worse than in cases of dropsy. In those cases in which both cerebral symptoms and dropsy exist the prognosis is quite bad.

LECTURE XLIX.

DISEASES OF THE KIDNEYS—CONTINUED.—CHRONIC PARENCHYMATOUS NEPHRITIS.—ACUTE DIFFUSE NEPHRITIS.

Chronic Parenchymatous Nephritis; Pathology, Etiology, Symptoms, Changes in the Urine, Dropsy, Duration and Course of the Disease, Treatment.—Acute Diffuse Nephritis; Synonyms, Pathology, Occurrence, Varieties of Idiopathic Cases, Symptoms of First Set of Idiopathic Cases, Prognosis.

CHRONIC PARENCHYMATOUS NEPHRITIS.—*Gentlemen:* Besides cases of acute parenchymatous nephritis we meet also with cases of chronic parenchymatous nephritis.

Pathology.—In these the lesion in the kidneys is of the same character as in acute parenchymatous nephritis. The morbid changes are confined to the epithelium of the tubes and to the epithelium lining the Malpighian bodies. We find, just as before, a swelling of these cells, a degeneration of some of them, a breaking down and desquamation of some, and, in addition to these changes, in some cases the epithelium is more markedly granular, even fatty, and fills many of the tubes. There may be hyaline cast matter in the tubes of the pyramids. There are no changes in the intertubular tissue.

Etiology.—A number of cases occur without any known cause. In others the condition follows acute parenchymatous nephritis; the patients, instead of getting well, continue to suffer from the same changes in the kidney and the same symptoms, the disease becoming chronic.

Symptoms, Changes in the Urine.—In every case we find changes in the urine. In a considerable number of case the specific gravity will remain at the normal, the patients passing urine which has a specific gravity of from 1,020 to 1,025; in some cases it will be a little above the normal, 1,030 to 1,035, and in still other cases it will be below the normal, from 1,015 to 1,017 or 1,020. There is nothing characteristic in the specific gravity of the urine, except that it does not differ very much from the normal. The quantity of urine also varies during a considerable part of the time. The patients may pass as much as healthy persons, but from time to time it will be diminished to ten to twenty ounces during the twenty-four hours; then it may again return to the normal. Albumen is usually present, but the quantity of this, again, varies during the course of the disease. There will be days and weeks when there will be fifty, sixty, seventy, or even eighty per cent. of albumen, then there will be days or weeks when it will be present in very small amounts. Casts are present as a rule, but in small numbers. A good many specimens of urine may be examined in which you will find no casts at all. The sort of casts which you do find are the hyaline, granular, and nucleated varieties.

Dropsy.—These patients, as a rule, develop dropsy, and when this occurs it is apt to be quite marked. There will be not only dropsy of the legs, but of the scrotum, and an accumulation of serum in the abdominal and pleuritic cavities.

There are very often disturbances of the stomach; patients lose their appetite, are troubled with nausea and vomiting, but these are commonly not very persistent or very troublesome. The patients vomit from time to time, but not constantly. The blood is apt to become changed—thin and watery, so that the color of the patients is altered; they become of an unnatural white color. The accumulation of serum in the pleuritic and peritoneal cavities, pressing upward, may give rise to a certain amount of dyspnœa, or shortness and difficulty of breathing, and it is even possible for them to develop œdema of the lungs. There are also cerebral symptoms; the patients become stupid, mildly delirious, and the stupor may even go on to semi-coma; but, except in fatal cases, the cerebral symptoms are usually not very marked. There is as a rule only a little stupor and perhaps mild delirium, but in all cases toward the close of the disease the patients may become more delirious, semi-comatose, and may even die comatose.

Duration and Course of the Disease.—The duration of the disease is considerable. It lasts for a number of months; but during this time there may be periods when the patient is better; the dropsy will diminish, the urine will become greater in quantity, and for weeks or months the patient will be quite well, after which time he will suffer as before. In a certain number of cases, after lasting in this way for a number of months, the patient will begin to improve, and this time the improvement may be permanent; the dropsy will go away, the appetite return, and the urine become normal in quantity; the condition of the patient will be normal, and he will remain in this state for an indefinite time. Apparently it is possible for these cases to recover completely; but in the cases that recover—as in those of recovery from acute parenchymatous nephritis—the changes in the urine persist for a long time. After the other symptoms have disappeared, you will find that it contains a little albumen. Again, the termination of the disease may not be as favorable as this. The patients, instead of getting better, continue to get worse; the dropsy persists, the disturbance of the stomach continues, cerebral symptoms become more marked, and the patients finally die dropsical, or with cerebral symptoms, or from some intercurrent disease, or exhausted from the disease itself.

Treatment.—The treatment of these cases is very much the same as those of acute parenchymatous nephritis, in which the dropsy is a prominent symptom. It does not do to be in too much of a hurry in trying to get rid of the dropsy. At first, you must keep the patient comfortable, put him on a milk diet, and, after waiting for a time, you should endeavor to get rid of the dropsy by means of the skin, bowels, and kidneys. If, after trying these different plans for a week, or two, or three weeks, you find that the dropsy remains the same, the urine is not increased in quantity, and the general condition of the patients is not improving, you will do well to stop their use, and leave the patients entirely alone, except that you give them the muriated tincture of iron, or any of the preparations of iron which you may prefer. After awhile you will try to get rid of the dropsy again, but there is no use of sweating, purging, or administering diuretics to these patients when you make no headway. It is better to stop, and simply endeavor to keep up the general nutrition of the patient, and, after waiting awhile, try again. In those cases that recover, after the dropsy has disappeared, the patients usually require the administration of iron for a considerable length of time, and, if possible, they should be sent to a warm climate for a time, especially during the succeeding winter; for it is known that, after they have been entirely well for months, they may have a recurrence of all the symptoms. More than this, the patients who suffer from parenchymatous nephritis may suffer from more than one attack. They may get entirely well of one attack, may return to their business, and be apparently well for five, six, eight, or ten years, during which time the urine may be entirely normal, and there will exist no reason for supposing that there is any disease of the kidneys, and yet, after this time, the patients may have a second attack, resembling, in all respects, the first. In these cases it seems that the kidneys do return to a normal condition, and that the patients suffer from the second attack in the same way as from the first.

ACUTE DIFFUSE NEPHRITIS.—We come now to acute diffuse nephritis.

Synonyms.—This lesion of the kidney you will find described in different text-books under different names. Some speak of it under the name of acute Bright's disease, others call it acute desquamative nephritis, others call it acute tubular nephritis, others, again, call it croupous nephritis, still others call it acute albuminuria; by some it is known as the first stage of chronic Bright's disease; by others, acute parenchymatous nephritis, and by others, acute interstitial nephritis. You will observe that the number of names employed by different authors is very considerable, and it is one of the causes which has led to confusion in appreciating what this condition is. Although the names are numerous the lesion of the kidney is constant.

Pathology.—In acute diffuse nephritis we find the kidneys increased in size, and this may be very marked, so that they will weigh sixteen, eighteen, or twenty ounces. The capsules are not adherent; they strip off readily, and the surface of the kidney will be found perfectly smooth. The cortical portion is thicker than it should be, and is of an opaque white color, or this opaque white color may be mottled with hemorrhagic spots, or parts of the cortex may be congested. The pyramids are congested, are large and red, and the veins are filled with blood. The mucous membrane lining the pelvis and calices of the kidney is intensely congested, and may be in some cases of a livid color; in other cases it is of a natural appearance. When we come to examne the minute structure of the kidney, we find that there are regular changes in the tubes, Malpighian bodies, and stroma. The lesions are not like those of parenchymatous nephritis, which are confined to the epithelium of the tubes and Malpighian bodies. The changes in the tubes involve all of them. In the cortex you find the epithelium of both the straight and convoluted tubes changed. The cells lining these tubes have become swollen, degenerated, and detached from the walls of the tube, so that the tubes are blocked up with these desquamated and degenerated epithelial cells. We also find cast matter both in the tubes of the pyramids and cortex. The changes in the epithelium of the tubes are, as a rule, more marked in acute diffuse nephritis than in parenchymatous nephritis The changes, you will observe, are of essentially the same character, but more marked. The production of cast matter is much greater. There are changes in the epithelial cells which line the capsules of the Malpighian bodies, but these are different from what we find in parenchymatous nephritis; in the latter affection they are simply swollen, so that we see them more plainly than in health. In acute diffuse nephritis they are increased in number, and this may be to a very marked extent, so that a considerable part of the cavity of the Malpighian bodies will be occupied by these new cells, which are exactly like the normal ones in this situation. They are flat, nucleated cells, but enormously increased in number, and as they increase in number the tufts of the blood-vessels will be compressed. The change in the stroma of the kidney consists in an infiltration of its substance with serum and pus-cells. The pus-cells may be present in very considerable numbers, but they never seem to be numerous enough to form an abscess, or to collect in one place to form an abscess; the process never fairly resembles suppurative nephritis. Besides this, we may find extravasations of blood in the Malpighian bodies, in the tubes and in the stroma of the kidney, and we will also find a swelling of the connective tissue-cells which are found normally in the stroma of the kidney. You observe, then, that there is a marked difference between lesion of parenchymatous and acute diffuse nephritis.

Occurrence.—This disease presents itself either idiopathically or as a complicating lesion of scarlet fever. When it occurs as an idiopathic disease it is quite frequently the result of exposure to cold. In a considerable number of cases the history is quite explicit upon this point, the patients having been exposed to cold, or cold and wet, after which the symptoms have developed. In other cases, no history of exposure to cold is obtainable.

Varieties of Acute Diffuse Nephritis.—Of the idiopathic cases of diffuse nephritis, we have to distinguish two varieties, which are different in their clinical symptoms, although the lesion of the kidney is the same.

Symptoms of First Set of Idiopathic Cases.—In the first set of cases the invasion of the disease is acute. An individual who has previously been in good health, but after exposure to cold, sometimes without exposure to cold, will suddenly be attacked by rigors, which are accompanied or followed by a febrile movement, and very soon the patient will suffer from well-defined pain in the back, referred to the region of the kidneys. He will also be troubled with frequent and painful micturition, the urine being passed in small quantities, sometimes only a few drops, so that there may be almost complete suppression, or the patient may pass no urine at all for a considerable length of time. The amount of urea in the urine is diminished. The appearance of the urine is also changed ; it is of a bloody, dark, smoky color, and when we examine it we find that it contains a considerable amount of albumen, becoming almost solid upon the application of heat and nitric acid. The microscopical examination of it reveals in it the existence of blood, renal epithelium, and casts in very large numbers. There is no form of kidney disease in which we find so many casts as in acute diffuse nephritis. They are either hyaline, granular, nucleated, or epithelial. After the disease has lasted for a little time, fatty casts will be found in the urine, and we may also find renal epithelium and sometimes pus-cells, the latter coming from the kidney or bladder, it is difficult to say which, on account of the cystitis which is apt to complicate the inflammation of the kidneys. The specific gravity of the urine is diminished, although the patients pass a very small quantity. It will vary from 1,010 to 1,020. The patients soon develop dropsy, which varies in extent considerably in different cases, and is apt to involve only the face, or the face, feet, and hands ; there may be general anasarca, and besides the exudation of serum into the subcutaneous connective tissue and serous cavities, it may also be exuded into the lungs and the patients will suffer from œdema of the lungs. There may be an exudation of serum into the connective tissue beneath the mucous membrane, in the upper part of the larnyx, giving rise to œdema of the glottis, or there may be considerable effusions of serum into the pleural, peritoneal, and pericardiac cavities. There are disturbances of the stomach. The patients lose their appetite. They may have nausea and frequent and persistent vomiting. As a rule, there are cerebral symptoms, the patients complaining of a headache, and they become unnaturally drowsy and stupid. The drowsiness and stupor may alternate with delirium. In the bad cases the patients have general convulsions, become fairly comatose, and remain in this condition until they die.

These are the regular symptoms of the disease, and they are developed with different degrees of severity in different cases according to the intensity of the inflammatory process in the kidney; if it be very intense, the symptoms become marked, the patients get constantly worse, and die at the end of a few days, either with convulsions or comatose. If the lesion of the kidneys is not developed to such an extent, the symptoms will be less severe, though most of them will be present. There are still other cases in which the symptoms are only moderately severe, but in which the duration of the disease is much longer, the patients continuing to live and to have the changes in the urine which have been mentioned, and general renal symptoms. They may continue to have these symptoms for months, and finally get well or die from the kidney disease. In still other cases, the disease, after continuing a certain length of time, will gradually change its character, and the patients will go on to suffer from the lesions and symptoms of chronic diffuse nephritis.

In a certain number of cases the course of the disease is still further modified by complicating inflammations. Patients who are suffering from an attack of acute, diffuse nephritis seem to be predisposed to inflammations of the pericardium, lungs, pleura, and peritoneum. These will come on while the kidney disease still exists, and the course of the latter will be modified and the symptoms changed by the occurrence of the complicating inflammations, whether it be pericarditis, pneumonia, pleurisy, or peritonitis.

Prognosis.—The prognosis of this form of inflammation of the kidney in a very considerable number of cases is good. Patients, the subjects of the milder forms of the disease, get well at the end of two or three weeks; those affected with the more severe form continue to be sick for a long time—for several weeks or months—after which they may get well. The severe cases end fatally at the end of a few days with cerebral symptoms, or they die after the lapse of several weeks or months from the direct effect of the continuance of the disease; others die from one or other of the complicating inflammations, and still others go on to suffer from chronic, diffuse nephritis.

LECTURE L.

DISEASES OF THE KIDNEYS—CONTINUED.—ACUTE DIFFUSE NEPHRITIS—CONCLUDED.

Second Set of Cases of Acute Idiopathic Diffuse Nephritis; Symptoms, Prognosis, Treatment of Both Sets of Idiopathic Cases.—Nephritis Complicating Scarlet Fever; Three Forms, Symptoms Peculiar to Each, General Symptoms, Prognosis, Treatment.

ACUTE DIFFUSE NEPHRITIS.—*Second Set of Idiopathic Cases.*—*Gentlemen:* We will take up to-day the subject of acute diffuse nephritis, at the point where we left off at the close of our last lecture. In the second set of cases of idiopathic acute diffuse nephritis the invasion of the disease is not as sudden as in the first, or rather, it is not as distinctly marked; it may come on pretty suddenly, but the symptoms are not as marked as in the first set of cases. The patients will not notice them for a little time, or they will pay but little attention to them, and for this reason patients will say that they can hardly tell when they begin to feel sick.

Symptoms.—The first symptom is usually some disturbance of the stomach. The patients lose their appetite, are troubled with nausea, and may occasionally vomit. They may also have some pain in the back, but this is not very intense and may be absent altogether. They suffer from a feeling of general malaise; they do not feel like attending to their ordinary work. Sometimes they do continue to work or attend to their ordinary business, but yet they are conscious of not feeling well. Then they notice a change in the urine; that they are passing a less quantity than they have been in the habit of doing. Some patients notice a change in the appearance of the urine; that it is of a reddish or brownish color. This change in the color of the urine is not always present. The urine may remain of a natural color although diminished in quantity, the patients continuing to pass from fifteen to twenty ounces; it is not suppressed. When you examine the urine you find that it contains a large amount of albumen and a great many casts, which are of all varieties, hyaline, granular, nucleated, and epithelial. There may also be a moderate number of blood-globules in the urine. Its specific gravity is lower than normal. The amount of urea contained therein is less than it should be. The patients usually develop a certain amount of dropsy, which makes its appearance, first in the face and then in the feet, or first in the feet and afterward in the face. The amount of dropsy varies in different cases, from a very little swelling of the face and feet to a condition of general anâsarca and dropsy of the serous cavities. The cerebral symptoms are not marked ; there may be a little headache and the patients may be a little duller than normal. There is a change in the composition of the blood ; it becomes thinner and more watery, as a consequence of which the mucous membranes are of a pale color and there is a certain loss of muscular strength, which is, as a rule, not very decided. The duration of these attacks is usually several weeks ; they may continue for several months.

Prognosis.—The prognosis is usually good, a considerable number of the patients recovering at the end of a few weeks or months. Some of them, instead of recovering, go on to suffer from chronic diffuse nephritis, the lesions in the kidney changing their character, and the patients suffering from symptoms which belong to this changed condition.

Treatment of Both Sets of Idiopathic Cases.—The treatment of these two different sets of cases varies somewhat. In the first set of cases, in which the invasion is acute, the constitutional disturbances well marked, and cerebral symptoms present, the indications seem to be in favor of tolerably active treatment. We put these patients to bed ; sweat them as profusely as we can with hot air-baths. We act upon the bowels by means of cathartics, and after the lapse of a few days, or a week or so, we begin the use of diuretics. When the function of the kidneys is returning to its natural condition and the general symptoms are beginning to abate, we diminish the use of these active measures and continue to give diuretics. I should have said, that, at the commencement of the disease, if the patients are strong and robust, you may apply wet cups over the region of the kidneys ; if they are not strong, you may apply dry cups, and these may be repeated for a number of days. When the patients begin to get better, you simply use diuretics to a moderate extent, keeping them upon a fluid diet, and when the function of the kidneys, as regards the amount of urine which is passed, is normal, you give up the use of diuretics, and commence the administration of one of the preparations of iron. After this you continue to keep the patients in the house until the urine contains no albumen and no casts, and until the weather is warm, when the patients may be allowed to go out of doors. Even though the urine contains no albumen and casts, it is important that these patients continue, if possible, in a warm climate for several months after recovering from the disease.

In those cases in which the symptoms are not as marked, in which there is no decided invasion of rigors and febrile movement, and the amount of urine, although diminished, is still considerable, when the general symptoms are not marked, the treatment does not need to be as energetic. A considerable number of patients will need nothing but to be kept in the house and confined to a milk diet. During convalescence you usually have to give iron. If the dropsy continues, you will have to aid its removal by means of the skin, bowels, and kidneys; sometimes moderate purgatives, after two or three days, will be sufficient to take away the dropsy; in other cases measures have to be kept up for a greater length of time. Like the other patients, they should be kept in a warm climate, that is, if possible.

NEPHRITIS COMPLICATING SCARLET FEVER.—We find that patients may suffer from three different conditions, which resemble each other to a certain extent but which are different in character. Some of the cases of scarlet fever are complicated by acute diffuse nephritis, other cases are complicated by parenchymatous nephritis, and in still other cases of scarlet fever the patients suffer from a moderate degree of dropsy of the subcutaneous connective tissue without any change in the kidneys whatever. These three conditions, which are distinct from each other, are often confounded, and it is sometimes very difficult to distinguish between them, but there is no doubt as to their existence as separate conditions. The fact that in the course of scarlet fever we have both acute, diffuse, and parenchymatous nephritis occurring has rendered it difficult to fix a period at which time the kidney lesion is most likely to develop. It is said that both may be developed from the very first day of scarlet fever to the end of the ninth week, that most cases begin on the fourteenth day, that next in frequency to this comes the twenty-first day, and next in frequency comes the seventh day. But these figures can only be taken as true of the two diseases taken together, both acute parenchymatous and acute diffuse nephritis. We have no tables as to the time when these lesions begin separately, but we have some reason for believing that parenchymatous nephritis begins usually earlier than acute diffuse nephritis. We know a considerable number of cases in which the changes in the urine in scarlet fever coming on early are due to parenchymatous changes, but still we have no very great statistics on this particular point. Both of these conditions of the kidney seem to be regular complications of scarlet fever. It is sometimes said that they occur as a result of exposures while suffering from scarlet fever, but in the larger number of cases no such exposure to cold can be made out. The kidney lesion seems to develop as something belonging to the regular history of scarlet fever.

In the cases of parenchymatous nephritis the symptoms are almost confined to the condition of the urine. This is diminished in amount. It contains albumen and a moderate number of casts, but, except for these changes, there are no symptoms whatever. There are a considerable number of patients in whom you would have no idea that there was any nephritis. It is only when you examine the urine and find it diminished in amount, and that it contains albumen and casts, that you know the patients are suffering from parenchymatous nephritis. Sometimes parenchymatous nephritis is more severe than this, and then there will be a little drowsiness and headache, and that will be all.

In acute diffuse nephritis which complicates scarlet fever we regularly find changes in the urine and also general symptoms. The changes in the urine are the same as those in idiopathic acute diffuse nephritis. The urine is diminished in quantity, is of low specific gravity, its color is changed by the admixture of blood, it contains less urea than normal and a large amount of albumen. Upon examination with a microscope we find red blood-globules and casts of all varieties.

General Symptoms.—Of the general symptoms we notice, in the first place, disturbances of the stomach. The patients lose their appetite, and may have some nausea and occasional vomiting. Usually there is a febrile movement, but, as a general thing, it is not very intense. Some patients complain of pain in the back, which may be quite severe, or of only moderate intensity; or, instead of the pain being confined to the back, a patient will complain of a general pain in the back and limbs. There will be cerebral symptoms, the patients may become unnaturally irritable, peevish, and complainful. They will suffer from headache, will be drowsy, the drowsiness may go on to stupor, and the stupor may give place to coma. The drowsiness will often alternate with delirium, the delirium may be very mild or quite severe. Occasionally, in children, there will be convulsions, which will be repeated a number of times. The bad cases will go on and the patients die in an attack of convulsions, or die comatose. You will also notice a change in the color of these patients; the blood becomes pale, and there is an unnatural pallor of the skin and mucous membrane. There will be dropsy, which may be moderate or very marked indeed; when it is marked it is usually general anasarca, involving the subcutaneous connective tissue throughout the body, and there may be collections of serum in the serous cavities, and œdema of the lungs.

The duration of these symptoms is usually not very considerable. They last for one, two, or three weeks, and then the patients gradually begin to improve; the general symptoms improve first, the condition of the urine improves more slowly.

Prognosis.—The prognosis of this condition is fairly good. The larger number of patients get well at the end of two or three weeks. In the more severe cases, however, the prognosis is not as good; the patients may die at the end of a few days with cerebral symptoms, or the symptoms of the disease may continue, and the patients may die at the end of a number of weeks with general dropsy or from general deterioration of the whole system; or they may go on to suffer from chronic diffuse nephritis, and the symptoms belonging to this.

Treatment.—The treatment of this condition does not usually require to be very energetic; the patients are not very sick. You simply keep them quiet in bed, and put them upon a milk diet.

Keep the bowels rather more open than ordinarily. You do not purge them. This will be all the treatment that is necessary in the mild cases. If, however, the symptoms are more pronounced, it may be necessary to use active purgatives, to apply cups over the region of the kidneys, and use hot-air baths, and the patient may require the administration of iron after recovering from the more severe symptoms; but most cases in scarlet fever do better if you do not treat them very energetically. The natural course of the disease is toward recovery at the end of a moderate length of time.

LECTURE LI.

DISEASES OF THE KIDNEYS—CONTINUED.—CHRONIC DIFFUSE NEPHRITIS.

Chronic Diffuse Nephritis; Synonyms, Pathology, Occurrence, Symptoms, Theories of Production of Dropsy, Dyspnœa, Loss of Vision, Neuralgic Pains, Cerebral Symptoms and Theories of their Causation, Changes in the Blood.

CHRONIC DIFFUSE NEPHRITIS.—*Gentlemen:* We go on to-day with the consideration of chronic diffuse nephritis.

Synonyms.—Like acute diffuse nephritis, this lesion of the kidneys is known by a very considerable number of names. It is described by some authors simply under the name of chronic Bright's disease; others speak of it as chronic croupous, catarrhal, or interstitial nephritis, or under the name of chronic tubular nephritis. Others simply speak of the kidneys as atrophied, granular, cirrhotic, or as the large white kidneys.

Pathology.—The lesions are very marked indeed, and involve nearly all of the different structures which make up the kidneys. The capsules are in many cases adherent to the surface of the kidneys, and when you attempt to strip them off you will tear up little pieces of the kidney tissue. The tubules undergo very decided changes, especially in the cortical portion, but also, though to a less extent, in the pyramidal portion. The epithelium of the tubes is changed. In some cases the epithelial cells are swollen, degenerated, and broken down into granular matter, or they may become detached from the walls of the tubes. In other cases, instead of this, the epithelial cells will remain in place. The cell-bodies will not be very much changed, but will be unnaturally flattened against the wall of the tube. Those in the convoluted tubes, instead of occupying a considerable part of the lumen of the tubes as they normally do, will be flattened against the wall of the tubes, so as to appear very thin. Besides the degenerated, altered, and compressed epithelium in the tubes, we find cast matter and, occasionally, globules of blood and fat, and sometimes pus-cells. The calibre of the tubes is often changed, The tubes may be dilated, and this may reach such an extent as to produce cysts, which can be seen with the naked eye. The dilatation of the tubes usually affects groups of them. There is apparently an obstruction of one or more of the straight tubes in the pyramidal portion of the kidney, and as a result of this, you will find in the cortical portion groups of dilated tubes. The group of tubes in the cortical portion of the kidney corresponds to one or more of the obstructed straight tubes in the pyramidal portion. Instead of this, the tubes may be atrophied ; the epithelium will disappear from their inner part, and they will become flattened so that you can hardly distinguish them from the ordinary connective tissue. The basement membrane also becomes changed ; sometimes only thickened. In other cases the wall of the tube is infiltrated with a waxy material or amyloid matter, and when this is the case the wall of the tube will also be somewhat thickened.

There is a production of permanent new connective tissue in the stroma of the kidneys, both in the cortical and pyramidal portions. In the cortical portion it is usually not diffuse, but occurs in circumscribed patches, so that if we make a section of the part we do not find new growths of connective tissue everywhere separating the tubes, but we find patches, larger or smaller according to the duration and development of the disease. These patches of loose connective tissue are formed of basement substance and small round cells. The Malpighian bodies are very frequently changed. The capsules may be thickened, contracted, or dilated. The epithelial cells which line the capsules may be simply swollen or increased in number. As in acute diffuse nephritis, the tuft of blood-vessels within the Malpighian bodies also become changed. The walls which make up this tuft may become thickened. The lumen of the vessels may become obliterated, and what was once a tuft of capillaries may be converted into a little hard body, through which the blood passes no longer. Or the walls of the blood-vessels making up the tuft may become the seat of waxy infiltration. The arteries are often changed. In some cases, the walls are hypertrophied, and at the same time, the lumen is diminished. In other cases, the walls of the arteries, especially of the smaller ones, become the seat of waxy infiltration. These changes, when they have once begun in the kidney, have a natural tendency to go on and become more and more marked. The disease is always developed slowly, and all these different changes begin in the kidney, at first to a very small extent, and then from month to month and from year to year they become more and more marked. There is a difference in the different kidneys as to the degree to which different parts of the lesion are developed. In one kidney the change will be more marked in the tubes, in another in the Malpighian bodies, in another in the stroma, and in still another in the arteries. Although we find this difference in the kidneys, yet we do not find that these lesions are developed successively, that there is first a lesion of the stroma, then a lesion of the tubes, or first a lesion of the tubes and then a lesion of the stroma, but in the very earliest examples of the disease with which we meet we find all these different changes present, the changes in the tubes, stroma, Malpighian bodies and vessels. When once developed, they go on to become more and more marked.

Although the lesion is essentially the same in all cases of chronic diffuse nephritis, yet the gross appearances differ considerably in different cases, and it is convenient to divide the kidneys according to their gross appearances into what are called large white kidneys, atrophied kidneys, and kidneys which are neither large white, nor atrophied: you may call these simply chronic diffuse nephritis.

In the large white kidneys the whole organ is very much increased in size: both the cortical and pyramidal portions are larger than normal. The capsules of these kidneys are usually not adherent to the surface, which, as a rule, is smooth. The cortex is most commonly of a uniform white color or white mottled with red, and the pyramids are of a red color.

The kidneys of old cases become smaller. They still remain of the same color, but the cortex is now thinner and the kidney is smaller than normal, instead of larger. Some of these atrophied kidneys are very small indeed. Sometimes both will only weigh three or four ounces. As a rule the capsules are closely adherent to the surface. When you strip off the capsule the surface of the kidney will be left nodular and uneven. Both the cortical and pyramidal portions of the kidney are atrophied, and it is especially in these atrophied kidneys that we are apt to find cysts which are large enough to be seen with the naked eye.

The kidneys which are neither the large white nor atrophied are sometimes of normal size and weight, sometimes a little larger than normal, or a little smaller. The capsules are a little adherent and the surface of the kidneys a little uneven. When we look at the kidney we do not see any marked changes in its gross appearance. The cortex and pyramids are of natural size and color. In other cases the color is somewhat changed, the normal striation of the cortical portion having disappeared. They may look so much like normal kidneys that you cannot be certain with the naked eye of any difference, and this may be the case when the lesion is so far developed as to have caused the death of the patient.

Occurrence.—Chronic diffuse nephritis is more common in males than in females. It occurs at nearly all ages, though not often in infancy. We do find examples of it in children and young persons, but the maximum liability seems to be in persons between forty-five and fifty-five years of age. It is a disease which prevails principally in temperate climates; it does not occur in tropical countries or in very cold countries. It occurs very frequently in individuals who have been habitually intemperate and in those who have been subject to privations of different kinds. It is a very frequent complication of cirrhosis of the liver, emphysema of the lungs, phthisis, and organic disease of the heart. It occurs not infrequently as a consecutive lesion in persons who have suffered somewhat from chronic inflammations of the joints or bones. It is somewhat commonly met with as a complication of syphilis and gout, and it may follow other forms of kidney disease; chronic congestion of the kidney, acute diffuse nephritis, and acute parenchymatous nephritis. There seems to be a disposition in certain families toward the development of the disease. I do not know that you can say that the disease is, strictly speaking, a hereditary one, but you will very often find in the same family a considerable number of examples of it. In a family consisting of a number of brothers and sisters, if one suffers from the disease you will very often find that other brothers or sisters of the same family will suffer from it, although you can hardly say it is transmitted from father to son. The disease also seems to have a certain relationship to chronic pulmonary phthisis; that is, in the same family in which phthisis is developed, other members of that family who do not suffer from phthisis will suffer from chronic diffuse nephritis.

Symptoms.—When the disease is fairly developed there are certain regular symptoms which are developed to a greater or less extent in different cases. In the first place, we expect to find changes in the urine. Its quantity is very apt to be altered. In some cases the patients pass too little urine, in others too much, and in others none at all for a number of hours or days. The specific gravity of the urine is regularly lower than normal, often very much lower. Unless there is some special reason to the contrary, the specific gravity is below 1,025, and very often below 1,010, or even 1,003. Its color is often paler or lighter than normal, and the patients also pass their urine more frequently than in health. When we come to examine its composition we find regularly urea in less quantities than in healthy urine. It contains albumen in varying quantities, from a very little to a good deal. In it are found casts, which are usually of the hyaline, granular, and nucleated varieties. Sometimes, however, we get fatty and epithelial casts. Another regular symptom of the disease is *dropsy*. This consists in the exudation of serum into the subcutaneous connective tissue of the face, feet, and legs. It is most commonly infiltrated in this way, but when the dropsy becomes marked, gradually the subcutaneous connective tissue throughout the body is infiltrated, so that the limbs and the body are swollen or made larger than they ought to be. The serum also accumulates in the different serous cavities, in the peritoneum, pleura, and pericardium, and also in the lungs, producing œdema of the lungs.

Theories of the Production of Dropsy.—It has always been a question as to how the dropsy is produced in cases of chronic diffuse nephritis. It is said by some that it is due to changes in the composition of the serum of the blood, that the serum is so altered by the disease that its specific gravity is made lower than normal, as a result of which it no longer remains in the vessels but transudes through their walls. Another reason which is given is that the constant passage of albumen from the kidneys into the urine changes the composition of the blood, and that, as a result of this constant loss of albumen on the part of the blood, the serum no longer remains in the vessels but transudes through their walls. Still another explanation which is given is, that a considerable number of these patients pass less urine than they ought, that they do not eliminate from their bodies the natural amount of water in the shape of fluid, and that for this reason the serum accumulates in the tissues of the bodies. Still another explanation is that the blood-vessels constantly contain more blood than they should, that they are always too full, but instead of being too full of natural blood they are too full of blood which contains an abnormal amount of serum and blood corpuscles, that it is not ordinary plethora but what is called hydroæmic plethora. Lastly, there is still one other explanation which is given, which is to the effect that there is a change in the composition or texture of the walls of the blood-vessels, and that in this way the serum of the blood escapes. These are the different explanations current at present as to the causation of dropsy.

Another set of symptoms which we find very often as a result of chronic diffuse nephritis are disturbances of the stomach. These are in some cases merely functional, not being accompanied with any actual lesion of the stomach. In other cases, they are associated with chronic catarrhal gastritis. The patients lose their appetite. They are apt to be troubled with nausea and vomiting, and besides, they may have some of the other symptoms of gastric dyspepsia; but loss of appetite, nausea and vomiting are very common and characteristic. The vomiting in many cases is exceedingly distressing and difficult to alleviate. It is very apt to be irregular. The patients will vomit one day and perhaps the next day will not vomit at all, and on the following day they will begin to vomit again, or they will go for several days without vomiting. Generally speaking, whenever the kidney disease is worse, the vomiting will be worse, and as the kidney disease becomes less marked the vomiting will likewise be less pronounced. The vomiting is usually excited by the ingestion of substances into the stomach. If the patients take food, or drink water, or take medicine, it will be very likely to set up vomiting, and when they have vomited what is in the stomach they may cease for a time and not vomit again until something is taken into the stomach. The irritability of the stomach in these cases is very great. Sometimes a teaspoonful of water will excite vomiting which will last for half an hour.

The function of the intestine is also interfered with. The bowels not infrequently become loose, especially when there is much dropsy—the looseness of the bowels seeming to be an effort on the part of nature to get rid of some of the fluid, the patients having, during the twenty-four hours, a number of loose, watery fecal passages.

Dyspnœa.—Very often there are symptoms referred to the lungs, the patients suffering from dyspnœa. The difficulty in breathing, however, seems to depend upon one or other of three causes or conditions. In some cases it is due to œdema of the lungs. There is an accumulation of serum in the lungs, and, in this way, their function is interfered with, the patients breathing with difficulty on account of a mechanical cause. In other cases, the dyspnœa is due to an accumulation of fluid in the pleural cavity; there is hydrothorax, either single or double, and the lungs are compressed by the fluid in the pleural cavities, so that they cannot perform their function. There is a third set of cases, in which there is neither œdema of the lungs nor hydrothorax, and yet the patients suffer from dyspnœa of a severe character. We have, as yet, no good explanation of the cause of this, although it is a common symptom of Bright's disease, and very characteristic. It is commonly said to be due to nervous influences; at all events, it occurs without any lesion of the lungs. This dyspnœa usually comes on in attacks like that of asthma, which it resembles very much, but there are no râles with it, and there is usually no sibilant or sonorous breathing. The patient moves all the muscles he can bring into play to get air into the lungs. Air does pass into the lungs, and yet the patient feels as if he were not breathing properly. These attacks of spasmodic dyspnœa are sometimes the first symptoms from which a patient with chronic diffuse nephritis suffers. An individual who has previously been in ordinary health will suddenly be attacked in this way with spasmodic dyspnœa, and he will go on to suffer from attacks of this kind for a number of months before any other symptoms of the disease are developed.

In a considerable number of cases of chronic diffuse nephritis, the patients lose their eyesight; they gradually become blind. The blindness is not usually total, but consists in the partial loss of vision. It regularly depends upon anatomical changes in the retina and optic disk; there may be either simple retinitis, or there may be the characteristic retinitis of chronic diffuse nephritis. In simple retinitis there is an infiltration of the retina with serum; as a result of this, the optic disk and the retina immediately surrounding it become swollen and opaque, and the patients no longer see as well as normally. The characteristic retinitis of Bright's disease, however, is different. There is not merely an infiltration of the retina and optic disk with serum, but parts of the retina undergo degenerative changes, so that the elements of the retina are destroyed and replaced by fat; and, as a result of this, the fundus of the eye becomes studded with opaque white spots, which represent the parts of the retina where the natural elements have been destroyed by this new matter. There are often extravasations of blood into the retina in these cases. These changes in the eye, again, may be one of the first symptoms of kidney disease. Those physicians who are familiar with eye diseases not infrequently have patients come to them complaining of loss of vision, or that their eyesight is failing. An examination of the eye shows the existence of either simple retinitis or nephritic retinitis. This is now so well known, that oculists, when finding this lesion, examine the urine, and inquire into the history of the patient, and in this way discover that patients who supposed they were simply suffering from loss of vision are in reality suffering from chronic diffuse nephritis.

Neuralgic Pains.—Still another symptom is the occurrence of neuralgic pains in different parts of the body. This symptom is, of course, not characteristic, for we meet with such pains in individuals due to a great many causes; but many patients who have chronic diffuse nephritis suffer from this symptom, and when you have under treatment a patient with neuralgic pains, and no cause can be assigned, you should examine the urine to see if there are any changes in it.

Cerebral Symptoms; Theories of Causation.—In addition, patients suffer regularly from cerebral symptoms—uræmic symptoms as they are ordinarily called. These are headache, drowsiness, stupor, delirium, coma, involuntary twitchings of different groups of voluntary muscles and general convulsions. Although these cerebral symptoms are common, we are still uncertain as to how they are really produced. One explanation which has been given is that there is an accumulation of urea in the blood, and in this way it becomes poisoned, giving rise to the cerebral symptoms. Another explanation is that the urea not only accumulates in the blood, but it becomes changed into carbonate of ammonia, and that the cerebral symptoms are due to poisoning, not by urea, but by carbonate of ammonia produced from it. Others believe that not merely urea accumulates in the blood, but that all the different substances which ought to be eliminated from the body through the urine accumulate in the blood, and these again interfere with the function of the brain. Still others think that the symptoms are due to changes in the brain, that it becomes unnaturally anæmic. These are the current explanations at the present time as to the causation of the cerebral symptoms, but none of them will answer.

Changes in the Blood.—Finally, we have changes in the blood. These are manifested by a change in the color of the patient. The patient becomes pale and anæmic. The skin becomes unnaturally white, and the mucous membranes unnaturally pale. The sclerotic coat of the eye is of an unnaturally white color, due to the diminution of the red blood-corpuscles and to the presence of serum in greater quantities than normally.

LECTURE LII.

DISEASES OF THE KIDNEYS—CONTINUED.—CHRONIC DIFFUSE NEPHRITIS—CONCLUDED.

Complications of Chronic Diffuse Nephritis; Heart Disease. Emphysema, Pneumonia, Apoplexy, Pericarditis, Peritonitis, Pleurisy, Cirrhosis of the Liver, Pulmonary Phthisis; Symptoms and Cause of the Disease; Symptoms of Large White Kidneys, Symptoms of Atrophied Kidneys, Symptoms of Kidneys neither Large White, nor Atrophied, Prognosis, Treatment.

CHRONIC DIFFUSE NEPHRITIS.—*Complications.*—*Gentlemen:* Besides the regular symptoms of chronic diffuse nephritis, we meet with certain complications of this disease, and these occur so frequently that we have to take them into account as part of the natural history of the disease.

Heart Disease.—The most frequent of these complications is heart disease. We find this associated with chronic diffuse nephritis in three different ways. In the first place, patients suffer originally from organic disease of the heart, either from valvular disease or dilatation of the heart, and this is followed by chronic congestion of the kidneys. This in turn is followed by the development of chronic diffuse nephritis, so that in these cases the kidney lesion is secondary to the heart disease. Then, in other cases, patients suffer first from chronic diffuse nephritis, and after a time they begin to have hypertrophy of the left ventricle of the heart. The cavity of the ventricle retains its usual size as a rule, but the walls become thicker. This is in the same way caused by the kidney lesion, though it is still uncertain how the hypertrophy of the left ventricle is produced. It is supposed by some that it is due to the obstruction of the circulation, to the passage of the arterial blood through the kidneys, and thus an increased amount of work is thrown upon the left ventricle. Others suppose that the urea which accumulates in the blood acts as a cardiac stimulant, and that in this way hypertrophy of the left ventricle is produced; that the heart is stimulated to an unnaturally forcible contraction.

There is still another set of cases, in which you can neither say that the heart or the kidney disease is primary. You find developed in the same patient organic disease of the heart and chronic diffuse nephritis, and you have to regard them as associated lesions, and as dependent one upon the other. This third set of cases is, in New York at least, the most common. The two lesions are developed in the same person, but you cannot say that either one is dependent upon the other.

Emphysema.—Of other complications, perhaps the most common is emphysema of the lungs. It occurs in a very considerable number of cases.

Pneumonia.—A fair proportion of patients with chronic diffuse nephritis suffer from ordinary lobar pneumonia. I do not know whether we can say that this is a complication of the disease; that is, whether the patients are any more apt to have pneumonia than other people, but if they do have it they are more apt to die than those persons whose kidneys have previously been healthy.

Meningitis.—Chronic meningitis does seem to be due in some way to the existence of the nephritis, for it is not usually developed until the kidney lesion has existed some time, and it occurs in connection with it more frequently than under other circumstances.

Apoplexy.—Apoplexy is another complication which seems to depend partly upon the kidney lesion and partly upon the associated heart lesion. These cases occur most frequently in those patients who have hypertrophy of the left ventricle of the heart as a result of kidney disease.

Pericarditis.—Pericarditis also seems to be due to the kidney disease. Acute pericarditis occurs very seldom as a primary lesion; it is found in nearly all cases in connection with rheumatism or pneumonia. If a patient has pericarditis, and has not rheumatism or pneumonia, I think the chances are nine in ten that he has Bright's disease or chronic diffuse nephritis as the cause of the pericarditis. Accordingly, you should examine the urine.

Peritonitis.—The same is true of peritonitis. Idiopathic peritonitis is a rare disease, though it occurs from time to time, and in those cases which we call idiopathic you should suspect chronic diffuse nephritis.

Pleurisy.—Pleurisy occurs frequently enough with chronic diffuse nephritis, but it seems to be associated with it in the same way as pneumonia. You can hardly say that these patients are more likely to have it than are other persons.

Cirrhosis of the Liver, Pulmonary Phthisis.—As associated lesions we have cirrhosis of the liver and chronic pulmonary phthisis.

Symptoms and Course of the Disease.—Although we have the regular symptoms which I have described to you and the complications of which I have spoken, yet you will find in practice that cases of chronic diffuse nephritis vary very much from each other. In some the diagnosis is made with great facility, in others with difficulty.

The course of the disease varies very much in different cases. We find that there is sufficient difference in the symptoms of the disease in patients who have large white kidneys and those who have atrophied kidneys to enable us to separate them into two sets, according to their clinical symptoms, and to this we may add a third set of patients, who have neither large white or atrophied kidneys.

Symptoms of Large White Kidneys.—The patients who have large white kidneys are those who develop the most pronounced symptoms and those in whom the diagnosis is most readily made. In these cases, as a rule, dropsy is developed early in the disease and in a considerable number of cases it will be the first symptom. When it has once begun it will continue, sometimes until the death of the patient; at other times it will come and go. In other cases, although dropsy is developed, it does not make its appearance at the commencement of the disease. The patients at first suffer simply from disturbances of the stomach. They lose flesh and strength. There may be a period of several months during which there will be no dropsy. The patients simply notice that they have lost their appetite; that they are troubled with nausea; that they vomit and have lost flesh and strength. After these symptoms have lasted for months dropsy develops. In still the third set of cases there will be no disturbance of the stomach, but there will be loss of flesh and strength and anæmia, and there will be neuralgic pains in different parts of the body. In all, after a few months, dropsy is developed. There are no cases, or, at least, there must be very few cases in which dropsy is not developed at some period of the disease.

In whichever of the three ways the disease begins, the patients get worse; some get worse continually until death; others have remissions. In the continuous cases the whole course of the disease is, as a rule, not very long. Patients die at the end of a few months, or a year, or two years from the time the first symptoms made their appearance. But there are patients in whom the course of the disease is not continuous. They will suffer from the different symptoms for a period of several months, then they will get better. The dropsy will diminish and disappear; the appetite return; the nausea and vomiting cease. They will begin to gain flesh and strength and become less anæmic. This period may last for one or two years and then the symptoms come on again in the same way and last for several months, and after a second attack the patients may again improve and return to a condition of apparent health. In this way the course of the disease may be protracted for a very considerable number of years, from five to seven or even from ten to twelve years, or longer than this, and during the whole of the time the kidney lesion will persist but the symptoms intermit.

During the entire course of the disease there are changes in the urine. It is usually diminished in quantity at the commencement and at the close: sometimes, toward the close of the disease, it will be suppressed. Its specific gravity is regularly lower than in health, but there will be times when it will be normal, or even increased. The urine always contains albumen and most commonly in very considerable quantities. It may become almost solid upon the application of heat and nitric acid. In it we find hyaline and granular casts in tolerable numbers. These changes in the urine make their appearance at the very commencement of the disease, and are of great help in making a diagnosis.

In those patients in whom there is at first no dropsy and the only symptoms are those of disturbances of the stomach, you will find very early in the urine these characteristic changes, and they usually persist during the intermissions of the disease, although the other symptoms disappear and the patient is apparently well.

The course of the disease is either intermittent or continuous, and the patients finally die. A certain number of them die apparently from the mechanical effects due to the accumulation of serum, joined with a general loss of nutrition. Others die suddenly and you can hardly tell why they do so. They will be suffering from the ordinary symptoms of the disease and while sitting up in bed or walking about will suddenly fall down and die, and upon post-mortem examination you will find no lesion to account for the sudden death. Still other patients die with cerebral symptoms. Toward the close of the disease they will become stupid, delirious, have repeated convulsions, and finally die in an attack of convulsions or comatose. Most patients die in one of these three ways.

Symptoms of Atrophied Kidneys.—When we come to symptoms of patients suffering from atrophied kidneys we find a much more difficult and obscure set of cases. In practice you will find a good deal of trouble in making out a diagnosis of many of these cases. In the first place, there is considerable difference in the way in which the disease comes on—in the first symptoms. It is evident from autopsies that patients suffer from this form of disease without manifesting any symptoms, for very frequently we find in the bodies of patients dead of some other disease that the kidneys are far advanced in chronic diffuse nephritis, and yet the previous history of the patient has indicated no kidney trouble. So we have to begin with admitting that patients may suffer from this form of nephritis for a considerable time without any symptoms due to the kidney lesion.

Then there is another set of cases which are of considerable clinical importance. Patients who have not given any kidney symptom at all and who become the subjects of some severe accident, who fall and break a leg, or sustain some severe wound, or acquire acute pneumonia or pleurisy, or become the subjects of any of the continued fevers will at once have kidney symptoms, although before this there was no reason for considering that the kidney was in any way affected. The kidney trouble existed and the accident was sufficient to bring out the symptoms. Again, other patients will at first simply suffer from occasional œdema of the feet. This will not reach any very marked extent. It will not be constant. Sometimes the feet will be a little swollen, sometimes not. Very often they are swollen at the end of the day and then in the morning, after the patient has been in bed all night, the œdema will have disappeared, and there may be days and weeks during which it will be absent altogether; then, it will again make its appearance. Besides this slight and varying œdema of the feet, these same patients will have headache, loss of appetite, occasional vomiting, and occasional attacks of spasmodic dyspnœa. These may be all the symptoms for some time.

In other cases the first symptom will be disturbance of the stomach. There will be no headache, but simply loss of appetite, a little nausea, perhaps vomiting from time to time, some anæmia and some loss of flesh and strength, but no œdema. These cases are very common indeed. One sees a great many examples of the disease developed in this way. The patients simply feel that they are not as well as they should be. They are rather paler than normal. Occasionally they have nausea, and any little article of food that disagrees with them will cause them to vomit.

Then there are other cases in which the first symptom will be loss of vision. The patients will simply notice that they are losing their eyesight, and it will not be until a considerable time after this that other symptoms will develop. There are other cases in which neuralgic pains will be the only symptoms, sometimes for months. In other patients the first symptoms will be an attack of convulsions, the patients having had nothing at all before this to call their attention to the condition of their general health. They will be suddenly seized with convulsions and die at the end of a few days, either in an attack of convulsions or comatose. There are still other cases which develop dropsy at the very first. They run a course very much the same as those with large white kidneys. They will begin with dropsy and this will continue. These are some of the ways in which the disease begins.

The urine in these patients indicates changes, but these are not nearly as constant or marked as in patients suffering from large white kidneys. The urine is, as a rule, passed in an increased amount. They not only pass too much urine, but they pass it too frequently. Toward the close of the disease, however, the urine is diminished in amount, and may even be suppressed, and during the course of the disease there will be periods of days or weeks when the amount of urine will be diminished, so that, though during the greater part of the disease it is increased, there are times when it is diminished or even suppressed. The urine is of low specific gravity, lower than in cases of large white kidneys, but this varies. It may be normal for months, though it is regularly very low. It contains albumen in small amounts, and the presence of albumen is very varied; it is sometimes present and sometimes absent. The urine contains casts, but these are also present in small numbers, and there will be periods when no casts are present. You will observe, then, that the urine in these cases of atrophied kidneys does not help in making a diagnosis in the same way as in patients having large white kidneys, for, at the particular time at which you see the patient, the urine may be of normal specific gravity. It may contain no albumen, no casts, and yet the patients may be suffering from advanced chronic diffuse nephritis, so that the fact of finding normal urine is nothing against the diagnosis, although, if you find changes in the urine it helps in making a diagnosis.

The course of the disease is usually very protracted. It is developed slowly and the patients continue to live for a number of years before they die. Either toward the close of the disease, or some time before its close, the patients are apt to suffer from attacks of cerebral symptoms. A considerable number of them die with delirium, convulsions, or coma, and these attacks may be developed from time to time during the course of the disease. You must remember, that you will see patients suffering from severe attacks of cerebral symptoms, and yet they will not die at that particular time. The patient will get better, and after this may live for a number of years. Occasionally, we see patients who suffer from a number of these attacks separated by considerable intervals, so that, although they are likely to die in such attacks, a considerable number of them do not. These patients may also develop a febrile movement at some period of the disease. This is especially the case at the time when they are suffering from cerebral symptoms. The thermometer may mark a rise of temperature, usually not over 99° or 100° or 101°. The temperature remains high for several days without any other lesion than that in the kidney to account for it. After a time these patients always end, as do those with large white kidneys, by dying ; some of them die with cerebral symptoms, some die dropsical, and others die suddenly, the mode of death being the same as with the large white kidney, and the prognosis is equally bad.

Symptoms of Kidneys neither Large White nor Atrophied.—In the third set of cases in which the kidneys are neither large white nor atrophied, the course of the disease is very much the same as those of atrophied kidneys. The only difference is that the changes in the urine are apt to be more marked. The albumen is present in larger amounts and is more constant, so that the diagnosis is made out more easily.

Prognosis.—The prognosis of all these forms of diffuse nephritis is the same. The natural course of the disease is progressive. The patients constantly become worse and worse as the kidney lesion increases, and after a longer or shorter time they die from the results of the disease.

Treatment.—The treatment of the disease is altogether a treatment of symptoms, the whole object being to make the patients as comfortable as possible and to prolong their lives. We endeavor to prolong their lives principally by treating the symptoms. If the patients are passing too little urine we try to increase the amount by the use of ordinary diuretics. If they are passing a considerable amount of urine we give them iron, with the idea of restricting its flow. If the urine contains albumen in large amounts we again give iron, with the idea of diminishing the amount. If there is dropsy we endeavor to get rid of it by means of the bowels, skin, and kidneys. If the functions of the stomach are disturbed we feed the patients in the best way we can; we put them upon a milk diet if they are unable to take solid food, or we ascertain what particular kinds of solid food they can take to the best advantage. Besides this, we use the ordinary remedies for dyspepsia to help the appetite and digestion. If the patients suffer from dyspnœa, and this is due to œdema of the lungs, we use dry cups over the chest. If it is of the spasmodic variety we use the same remedies as in spasmodic asthma. Unfortunately, they are much less efficacious in controlling the dyspnœa of Bright's disease than in asthma. Spasmodic dyspnœa is perhaps the most intractable of all the symptoms of chronic Bright's disease. So far as the conditions of the eyes are concerned, there is nothing to be done. Either of the forms of retinitis is not amenable to treatment. The neuralgic pains usually have to be treated with considerable doses of quinine or morphine, the quinine is preferable. The cerebral symptoms are treated in one of two ways, either by what is called the eliminative plan or by the administration of morphine. The eliminative plan of treatment consists in purging the patients, sweating them profusely by hot air baths, and acting upon the skin, bowels, and salivary glands, and by means of jaborandi. The other plan is to give hypodermic injections of morphine. Patients with kidney disease are also usually poisoned with opium, but at the time they are suffering from cerebral symptoms you can give them considerable doses without poisoning, and thus relieve them.

LECTURE LIII.

DISEASES OF THE KIDNEYS—CONCLUDED.—SUPPURATIVE AND TUBERCULAR NEPHRITIS.—CANCER OF THE KIDNEY.

Suppurative Nephritis; Pathology, Occurrence, Symptoms, Prognosis, Treatment.—Tubercular Nephritis; Pathology, Duration, Symptoms.—Cancer of the Kidney; Pathology, Symptoms, Treatment.

GENTLEMEN: The forms of nephritis which we have been considering so far have all been characterized by the presence of albumen and casts in the urine; there are still two other forms of inflammation of the kidney, in which there are no casts and no albumen in the urine. These are suppurative nephritis and tubercular nephritis.

SUPPURATIVE NEPHRITIS.—*Pathology.*—In suppurative or pyelonephritis, as it is often called, we find both the pelvis of the kidney and the kidney tissue are the seat of the inflammatory process. The whole kidney is increased in size; it is congested, and the congestion involves the mucous membrane of the pelvis, as well as the substance of the kidney. The surface of the pelvis is covered with pus, and scattered through the substance of the kidney we find little abscesses of different sizes; some are so small that you can hardly see them without the microscope; others are so large as to involve a considerable part of the kidney, and between these two extremes there are abscesses of many sizes; in other cases the pus, instead of being arranged so as to form abscesses, will be diffusely infiltrated throughout the kidney, but in all cases it is formed between the tubules of the kidney. The tubules undergo change only as a secondary result of the suppurative process. When the pus becomes so abundant as to encroach upon them, and deprive them of their vascular supply, they die, break down and degenerate, and in this way cavities are formed, which are filled partly with pus-cells and partly with the remnants of kidney-tissue. The lesion usually involves both kidneys at the same time, although it is possible for only one kidney to be implicated.

Occurrence.—This lesion of the kidney is almost always secondary to a previous inflammation of the bladder—to cystitis; and it is one of the most dangerous of the complications of cystitis. The forms of cystitis with which suppurative nephritis is most frequently associated, are that which is due to stone in the bladder, that which accompanies strictures of the urethra, or enlarged prostates of old persons, cystitis which accompanies paraplegia, less frequently the cystitis which occurs with gonorrhœa, and also that which is due to operations upon the bladder and urethra.

Symptoms.—When the disease occurs from cystitis due to stone in the bladder, strictures in the urethra, with paraplegia, and from operations upon the bladder and urethra, the symptoms are essentially the same. The patient, having previously had the ordinary symptoms of cystitis from which he has been suffering, will be attacked with rigors, and these will be repeated a number of times. The rigors will be followed by a febrile movement, and this in turn will be followed by a pretty profuse sweating; at the same time there is a rapid change in the appearance of the patient, and he may rapidly become very much prostrated, have a rapid and feeble pulse, anxious and drawn face, dry and coated tongue, and pass rapidly into a typhoid condition. These patients resemble sometimes persons in a typhoid state, at other times persons suffering from pyæma; but in all, the depression of the general condition is very marked and takes place very rapidly.

The urine is diminished in amount—it may be suppressed altogether; in it we find blood, pus, and mucus; the pus and mucus belong to the cystitis, and have usually existed in the urine, before the time of the attack of the suppurative nephritis. The blood seems to come partly from the bladder, and partly from the pelves of the kidneys. In this condition the patients rapidly become worse for several days, at the end of which they usually die from the disease.

When the disease occurs as a complication of gonorrhœa, it does so more frequently in the female than in the male. In every case the inflammatory process extends first to the bladder and there sets up an acute cystitis, and you have the symptoms of this, added to the previous symptoms of gonorrhœa. From the bladder the inflammation extends up along the ureters to the kidneys, where a suppurative inflammation is set up. When this occurs the patients have the same symptoms which I have just mentioned: they have rigors, a febrile movement, sweating, a rapid prostration, and death after a few days.

When this condition of the kidneys exists, if the cystitis is due to enlarged prostate, the symptoms are somewhat different. The cases of enlarged prostate occur, as you know, usually in old men; they experience a difficulty in passing their urine, and from time to time they suffer from attacks of cystitis, of greater or less severity. The way in which you will usually observe these cases will be somewhat as follows: the old man who has been suffering for years from difficulty in passing water and occasional cystitis will at some time find that he is again unable to pass his water; he will endeavor to do so, but will pass none whatever. Very frequently these patients have already learned to pass the catheter, on account of their liability to have these attacks. The patient, finding that he cannot pass his water, will pass the catheter as usual, but with a little more difficulty than he has before experienced; and, instead of drawing off much water, none at all comes away; in place of this there is a little blood. He then sends for his physician, who, upon arriving, repeats the introduction of the catheter into the bladder without much difficulty, but finds that only very little urine comes away, and with it there is mucus and a considerable amount of blood. The next thing will be for the patient to become very much prostrated. There are usually no rigors, and there may be no febrile movement. He does not feel able to go out of the house or the room, but remains in his room, either in bed or lying upon the sofa. During the next twenty-four hours after the commencement of the attack, the patient will pass a little urine, or rather, a little urine will be drawn off with the catheter; there will be much less urine than normal, and it will contain a little blood; during this twenty-four hours the patient's pulse is becoming more rapid and feeble, he does not feel like taking food, and becomes still weaker. After this, during a few days, the condition of the urine will continue the same—it will be passed or drawn off in small quantities, and contain blood; the prostration becomes more and more marked, the pulse more rapid and feeble, the skin cold, the patient's face anxious, and at the end of a few days he will die in a condition of collapse, with cold skin, and with rapid and febrile pulse.

Prognosis.—The lesion is a very fatal one; so far as I know, patients die from it at the end of a few days. It is seldom protracted for as long as two weeks, though occasionally patients live as long as this.

Treatment.—The treatment for these cases, so far as the disease is concerned, is futile. We can do nothing for the relief of the suppurative nephritis. The most important point is to prevent patients from acquiring such a lesion of the kidneys, and this is to be done by attention to the cystitis, for if patients do not have cystitis, or if it is soon relieved, they will not have suppurative nephritis. The moment a patient develops cystitis under any of the circumstances I have mentioned, it must be remembered that he is in constant danger of having complicating suppurative nephritis; hence, the cystitis should be treated as early as possible, and every pains taken to keep the condition of the bladder as nearly normal as you can.

TUBERCULAR NEPHRITIS.—The other form of kidney inflammation which we have to consider is tubercular nephritis. This usually involves only one kidney; it is comparatively seldom that both kidneys are affected.

Pathology.—The inflammatory process begins in the mucous membrane of the kidney, extends from this to the apices of the pyramids, and then upward through the pyramids to the cortical portion of the kidney. The inflammatory process results in a production of tubercular tissue, of pus, and new connective tissue, and these three products all have a tendency to very rapidly undergo what is called cheesy degeneration, so that after the process has lasted for some time, we find that a considerable part of the kidney has been destroyed, and that it is replaced by a mass of yellow cheesy matter. If the patient lives long enough the entire kidney may be destroyed in this way; as a rule, however, the kidney is destroyed somewhat symmetrically—it is hollowed out in cavities, each cavity corresponding to a pyramid. So that you will have the capsule of the kidney left, and instead of this there will be a number of cavities separated from each other by remaining portions of the kidney tissue. These cavities will be filled partly with cheesy matters and partly with pus. When this condition occurs in the kidney we are very apt to find a similar condition in the ureter belonging to that kidney, and in the bladder. If it is a male, the same condition is likely to exist in the seminal vesicle, prostate, and testicle; if it is a female, a similar lesion will probably exist in one or both of the Fallopian tubes, and in the body of the uterus. In either the male or female we find the same inflammatory process of the ureters and the bladder.

Duration.—The disease is usually a very chronic one; it lasts for a number of years. After it has existed for some time, changes usually occur in the other kidney, but these are of an entirely different character. The other kidney becomes the seat of chronic diffuse nephritis, with waxy infiltration of the Malpighian tufts and small arteries, so that the patient finally suffers from two distinct kidney lesions, and has the symptoms belonging to both conditions.

Symptoms.—At the commencement of the disease the symptoms are usually quite obscure. The patient will, perhaps, complain of pain over the region of the affected kidney, the amount of pain varying, sometimes being very severe and at other times amounting only to a feeling of discomfort in the position of the affected kidney.

These pains will continue, and the patient's general health will begin to suffer; he will lose flesh and strength, will feel that he is not as well as he should be, and yet there will be nothing definite to account for this general loss of health, flesh, and strength. After a time there will begin to be changes in the urine. The patients will pass from time to time urine which contains blood, pus, and amorphous granular matter; but still you will very likely be unable to say whether this condition of the urine is due to cystitis or tubercular nephritis, for there will be no local symptoms so far as the kidney is concerned, except the indefinite pains in the region of the kidney. In this way the patients may go on for a number of months, and these be the only symptoms. If the patient is a male, after the disease has lasted for some months in this way, a similar condition may develop in the prostate, testicle, and seminal vesicle. When the testicle is thus involved you will have the so-called tubercular testicle, and this will help you some in making a diagnosis of the patient's condition; for if a patient, having the symptoms I have already enumerated, develops a tubercular testicle, you may be pretty sure that he has suffered and is now suffering from tubercular nephritis. In some cases, after several months, the affected kidney will become enlarged to such an extent that you can appreciate it as a tumor in the abdominal cavity. When you find this condition, with a previous history to guide you, the diagnosis can usually be made with certainty; you can usually be sure that the patient is suffering from tubercular nephritis. If the tumor is large enough and sufficiently near the abdominal wall, you can introduce the needle of an aspirator with the idea of finding pus. If it is a case of tubercular nephritis, whether you get pus or not will depend upon the size of the kidney and whether the needle penetrates one of these small abscesses. If the cavities in the kidneys are large enough, and the needle happens to get into one of them instead of into the kidney tissue, you will get a little pus, and only a very little; but it helps in making the diagnosis between this condition and dilatation of the pelvis of the kidney—hydronephrosis. In hydronephrosis, if you aspirate and get the needle into the pelvis of the kidney and obtain pus, it will be in considerable quantities; hence, if upon aspiration of a kidney you draw off a very little pus, the kidney is probably tubercular, but if a considerable quantity of pus is drawn off, it is probably a case of dilatation of the pelvis of the kidney.

The next thing for the patient to develop will be the symptoms which belong to the chronic diffuse nephritis of the other kidney. The urine will now begin to contain albumen and casts in addition to pus and blood, which were in it before. The patient becomes more and more anæmic, the health fails rapidly, there is drowsiness of greater or less extent, and the patient finally dies in very much the same condition as one suffering from chronic diffuse nephritis.

Treatment.—The treatment of this condition does not amount to a great deal. The only treatment which seems to offer any chance of success is one that is not very frequently adopted, viz.: the extirpation of the affected kidney. In these cases the patients seem to die, not in consequence of the tubercular kidney, but from the secondary implication of the other kidney. Hence, if the diagnosis can be made out sufficiently early, and the tubercular kidney removed, it would seem to offer a chance for the patient to live. This operation has been performed, but so far as I know, never until after the secondary disease has been developed in the other kidney, so that the patients were not relieved.

CANCER OF THE KIDNEY.—There is still one other condition of the kidney which we have to consider, and that is cancer of the kidney—carcinoma of the kidney. Primary carcinoma usually involves only one kidney, and, for some reason, it affects the right much more frequently than the left. The new growth begins in the substance of the kidney, and usually grows very rapidly. It is, as a rule, the soft, hemorrhagic form of carcinoma, abundantly supplied with vessels, and grows very rapidly indeed, forming a tumor of considerable size in the region of the kidney. These new growths are developed much more frequently in children than in adults, and in children under ten years of age.

Symptoms.—The symptoms which belong to this condition are the formation of a tumor in the vicinity of the kidney, which grows forward and upward. The tumor is situated behind the intestine, and as it becomes larger and larger it pushes the intestine before it, until finally, when it becomes very large, the intestine may be pushed to one side, and through the anterior abdominal wall you may come directly upon the tumor; when it is not very large, you may feel first the intestine and then the tumor. Besides the tumor, there is also pain which is referred to the region of the affected kidney; it is like the pain of other cancerous tumors, being more severe in some individuals than in others. It is possible for it to be absent altogether, but it is generally present and is pretty severe. There are, as a rule, changes in the urine; the patients pass blood with the urine and most frequently in considerable quantities. There may be so much blood that it will be coagulated in the bladder, and part of it come away in the form of clots, and part in a fluid form. The bleeding is not continuous, but intermittent. Sometimes the urine will be clear, at other times it will contain blood in large amounts, and the patients soon begin to emaciate, lose strength, and run down, and at the end of a year from the time when the disease was first made out they commonly die.

Treatment.—In these cases of cancer of the kidney the one thing you have to do is to make the diagnosis. Of course, there is no more treatment for this than for cancer occurring elsewhere. You simply keep the patient comfortable with opium.

Diagnosis.—The important point is to make out the diagnosis, and in doing this you are guided by the age of patient, by the situation of the tumor, and the presence of blood in the urine. When you find a child under ten years of age with a tumor growing rapidly in the position of one of the kidneys, and passing blood from time to time, and beginning to emaciate and run down, there is usually no difficulty in making the diagnosis. If you see one of these patients while the tumor is still small, it usually grows so rapidly, and the abdomen in these children is of such moderate dimensions, that you can make it out very readily. In adults the diagnosis is much more difficult, for in them the abdominal cavity is large, and if the patients have a thick abdominal wall and the intestine is distended with gas, you may have a good deal of difficulty in making the diagnosis. These patients will complain of pain in the position of the affected kidney, will pass blood with their urine from time to time, and will suffer from loss of general health, and yet you will not be able to make out any tumor in the position of the kidney, and so long as this is the case the diagnosis remains uncertain. The diagnosis usually lies between cancer of the bladder and cancer of the kidney; so long as you cannot feel a tumor the diagnosis is uncertain. When the tumor becomes larger, then there is another difficulty in making the diagnosis, and that is that adults suffer from tumors of the abdominal cavity which in shape and position resemble cancerous growths of the kidney. These tumors are sarcomata, growing behind the peritoneum, and as they grow pushing the peritoneum and abdominal viscera before them. They not infrequently develop in the position of the kidney, and grow forward as do cancerous growths of the kidney. When you feel them through the abdominal wall, they impart the same feeling as does cancer of the kidney; not only this, but they will grow often as rapidly as cancer of the kidney. But in these retro-peritoneal sarcomata the urine does not contain blood, and it remains in its natural state, which helps us in making a diagnosis between these two conditions.

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